The Journal of HEREDITY

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Linkage in Habrobracon

The Tailless Rat

VOLUME 33 FEBRUARY, 1942 NUMBER 2

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The JOURNAL OF HEREDITY is published monthly by the AMERICAN GENETIC ASSOCIATION at 32nd Street and Elm Avenue, Baltimore, Md., and Victor Building, Washington, D. C. Address all communications regarding editorial matters to Room 308, Victor Building, Washington, D. C. Communications regarding the business management of The JOURNAL OF HEREDITY may be addressed to the publication office, 32nd Street and Elm Avenue, Baltimore, Md., or to the Victor Building, Washington, D. C. Entered as second-class matter February 24, 1923, at the postoffice at Baltimore, Maryland. Acceptance for mailing at the special rate of postage provided for in section 1103, Act of October 3, 1917, authorized December 20, 1918.

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The

Journal of Heredity

Vol. XXXIII **FEBRUARY, 1942** **No. 2**

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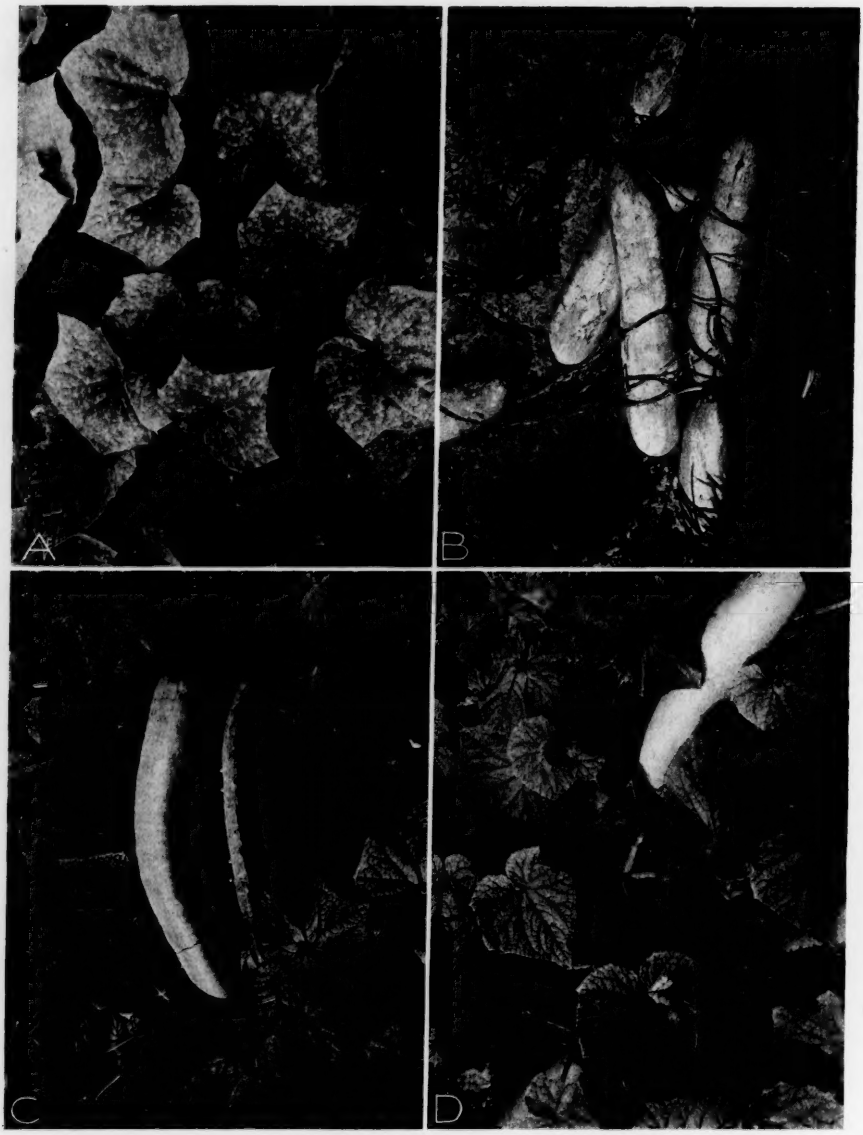


EFFECTS OF MILDEW ON SUSCEPTIBLE AND RESISTANT CUCUMBERS

Figure 1

The powdery mildew destroys susceptible varieties with remarkable rapidity. *A*—shows a plant of a susceptible commercial variety on June 27, the leaves being spotted with areas of mildew infection. On July 21, less than four weeks later, all leaves have been destroyed (*B*). A comparable plant of the resistant variety, China, is shown at *C* on June 27, and a resistant F_3 segregate of a cross of Colorado × Puerto Rico on July 21 is shown at *D*. This was grown under exposed conditions so that the comparison between *B* and *D* is a fair indication of the differences.

DOWNY MILDEW RESISTANCE IN CUCUMBERS*

J. Mitchell Jenkins, Jr.
South Carolina Truck Experiment Station, Charleston, S. C.

Downy mildew, caused by *Peronoplasmopara cubensis* (B. & C.) Clinton, is a very common and destructive disease of cucumbers in the South as well as in many other sections of the United States. In South Carolina it attacks the foliage of plants early in the spring soon after they begin to set fruit and defoliates them, causing serious reductions in yield. Cucumbers planted in the late spring or in the summer seldom produce satisfactory crops because of the prevalence of the disease.

The mildew fungus does not survive from year to year in the soil and is not carried on the seed. According to Doran[3] there is no evidence that oospores or other over-wintering stages of the fungus have been found, although several investigators have looked for them. Available evidence indicates that the fungus overwinters on live host plants along the Gulf Coast and in Florida and moves progressively northward in the spring following the planting of cucumbers. The spores are thought to be disseminated by the wind.

Various sprays and dusts have been recommended for the control of downy mildew. Nusbaum[6], working in South Carolina, found that the disease may be controlled by careful application of certain copper dusts. Although this method of control is satisfactory, it involves considerable labor and expense. Furthermore it may be relatively ineffective if the applications are not made carefully and at the proper times. It appears that the development of commercial varieties of cucumbers having resistance to downy mildew might offer a cheaper and more positive method of control than the use of fungicides.

Clayton[1], Weber[7], and Nusbaum[6] reported that all of the varieties of cucumbers tested by them appeared to be equally susceptible to downy mildew, but Kock and Kornauth[5] found that the varieties of climbing cucumbers were quite resistant. Cochran[2] reported that in Louisiana an Indian variety named Bangalore showed considerable resistance to the fungus. He made crosses between Bangalore and several slicing and pickling cucumbers and found that the F_1 generation was more resistant than the commercial parents. The F_2 segregated for resistance and some of the segregates showed as much resistance as Bangalore.

In 1939, to determine whether any available varieties or strains of cucumbers were resistant to downy mildew under South Carolina conditions, more than eighty lots of seed from various sources were planted in replicated blocks at the South Carolina Truck Experiment Station. Examinations at regular intervals were made to determine whether plants in the various lots exhibited differences in the degree of infection. Artificial inoculations were not attempted because, over a period of five years, it had been observed that all of the individuals in plantings of susceptible varieties became infected and died. No escapes had been found in any of the experimental plantings or in commercial fields.

Downy mildew appeared soon after the plants began to set fruit and within two weeks all of the commercial varieties were dead. It was found, however, that several of the less common varieties, mainly those from India and China, exhibited varying degrees of resistance. In Table I the relative resistance of twelve of the varieties is indicated and

*Technical Contribution No. 83 from the South Carolina Agricultural Experiment Station.

some of their other characters are listed.

The most resistant varieties were China and Puerto Rico No. 37, neither of which could be classed as a commercial type of cucumber. China is rather late and produces long, slender fruits which are pale green in color. Puerto Rico No. 37 is as early as Colorado, a standard variety, but it produces short, cylindrical fruits which are light green in color. Figure 2 shows the types of fruit produced by China and Puerto Rico No. 37 as well as the F_1 fruit from a cross between the two.

China and Puerto Rico No. 37 were compared with susceptible commercial varieties again in 1941. Before downy mildew appeared in Charleston County on June 19 all of the varieties had produced mature fruit and were wilting badly from drouth. Heavy rains fell at this time and the plants began to grow vigorously. Most varieties set more fruit. Conditions were very good for comparing varieties because all of them had produced mature fruit before becoming infected with the fungus. Differences in resistance could not well be attributed to differences in degree of maturity. Furthermore, Puerto Rico No. 37 continued to grow for three weeks after most of the Colorado plants were dead although it is just as early as the commercial varieties.

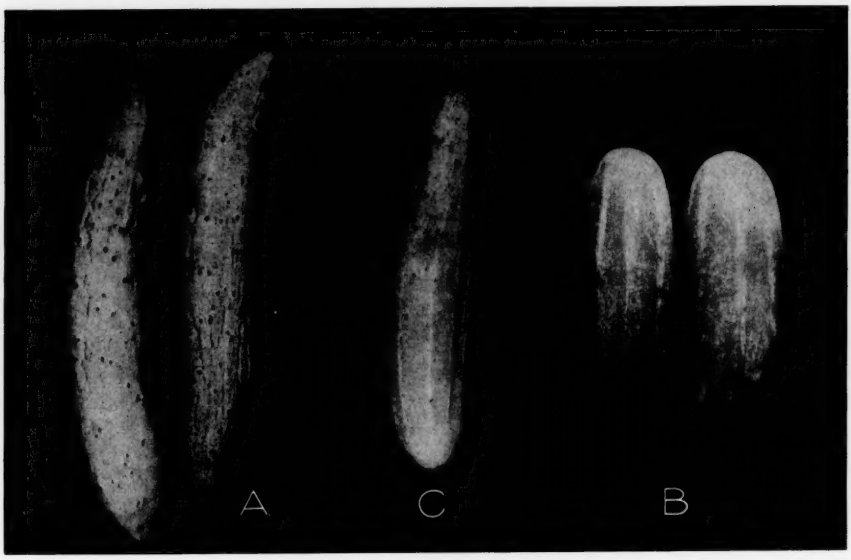
To facilitate a more accurate classification of the degree of downy mildew infection, counts were made of the number of lesions per leaf on each of the varieties soon after the first lesions appeared and again three days later. Following the first two counts plants were classified at intervals during June and July according to the amount of infection. An index of zero to 10 was used with 10 indicating complete susceptibility while zero indicated complete absence of visible mildew lesions. An index rating of 1 was given where minute lesions were found which did not appear to be spreading or enlarging, and a rating of 7 indicated that plants were so badly infected that necrotic areas were appearing. The data for four varieties are given in Table II.

It may be seen from Table II that there was a great difference in the degree of infection among the varieties. There were also differences in the rate of spread of the fungus on different varieties. Lesions on the leaves of Colorado or A. & C. enlarged rapidly causing the leaves to become yellow and die. Lesions on the leaves of China and Puerto Rico No. 37, however, remained small. The leaves of these two varieties seemed to die eventually as a result of the accumulation of large numbers of points of infection rather than from the enlarging of individual lesions. The fungus appeared to be inhibited and did not, at first, produce conidia on the under sides of the leaves of the two resistant varieties. Conidia were much more abundant on leaves of A. & C. and Colorado than on the resistant varieties all during the season and this was especially true for the two weeks after the disease first appeared.

TABLE I.—Comparison of 12 Varieties of Cucumbers Grown at Charleston, S. C., in 1939.

Variety	Downy mildew index*	Season	Fruit color and shape	Yield
A. & C. (Abbott and Cobb)	10	Medium early	Very good	Good
Straight-8	10	Medium early	Good	Good
Colorado	10	Medium early	Very good	Good
Japanese Climbing	10	Medium early	Fair	Poor
Mincu	10	Early	Fair	Poor
Spot Resisting	10	Late	Good	Poor
Palla	7	Late	Poor	Poor
Indian Telegraph	7	Late	Poor	Poor
Nepaul	5	Late	Poor	Poor
Bangalore	5	Late	Poor	Poor
China	3	Late	Poor	Poor
Puerto Rico No. 37	1	Medium early	Fair	Poor

*Varieties were classified on a scale of 0 to 10 with 10 indicating complete susceptibility to downy mildew and 0 indicating immunity.



MILDEW-RESISTANT VARIETIES

Figure 2

A—China; *B*—Puerto Rico No. 37; *C*—F_1 China × P. R. No. 37. The production of desirable commercial strains of resistant cucumbers involves the crossing of these varieties, or of hybrids between them, with commercial varieties. The resistant segregates, having the most desirable characteristics are used for further breeding and selection.

Practically all of the plants of the susceptible varieties were dead by July 7. The mildew index for China on this date was 5.1 and for Puerto Rico No. 37 it was 1.0. Certain individuals in both the China and the Puerto Rico No. 37 inbred lines showed greater resistance than the rest of the plants in the same lines, but many of the plants of China finally succumbed to downy mildew from two to three weeks after all of the plants of A & C. were dead. Puerto Rico No. 37 was less affected by the fungus than any other variety and seems to offer one of the best sources of genes for resistance to downy mildew.

TABLE II.—Comparative Degree of Mildew Infection of Four Cucumber Varieties at the South Carolina Truck Experiment Station, Charleston, S. C., in 1941.

Variety	Average number of lesions per leaf		Downy mildew index			
	June 24	June 27	June 30	July 7	July 15	July 21
A. & C.	11.20	30.4	8.5	10.0	10.0	10.0
Colorado	4.34	19.1	7.1	9.1	10.0	10.0
China	0.01	0.5	1.0	5.1	7.1	7.9
Puerto Rico No. 37	0.00	0.0	0.9	1.0	5.2	6.1

Crosses were made in 1939 between Puerto Rico No. 37 and Colorado and between China and Colorado for the purpose of combining resistance with the fruit color and shape of Colorado. Crosses were also made between the two resistant varieties, and in 1940 backcrosses were made to the three varieties. Most of the F_2 and backcross generations were grown in the spring of 1941 but a few were grown in the fall of 1940.

The F_1 plants from crosses between resistant and susceptible varieties were intermediate in degree of resistance. They were also intermediate in fruit shape and size. In the F_2, 17 plants were as resistant as the resistant parent, 26 were as susceptible as the commercial parent, and 545 were classed as intermediate. The intermediate plants,

however, ranged from those that were slightly less susceptible than the susceptible parent to those that were almost as resistant as the resistant parent. Since it is hard to classify individual plants for actual degree of resistance to downy mildew no attempt was made to work out ratios in the F_2. Instead, an F_3 population is being grown for a genetic study of the inheritance of resistance to downy mildew. The results of this study will be given in another paper.

Four resistant F_2 plants were very prolific and produced fruits of good commercial type, but the immature fruit color was poor. This was also true of several F_3 lines which were grown at the same time. Only three plants having fruits as dark green in color as Colorado were isolated from an F_2 population of 588 plants which seems to indicate that immature fruit color may be governed by more than one factor. If this is true it will be necessary to grow large populations in order to obtain resistant plants having as good fruit shape and color as the best commercial varieties.

A cross between China and Puerto Rico No. 37, both resistant to downy mildew, gave F_2 plants which were all resistant in varying degrees. None of these plants were as susceptible as Colorado or A. & C. and five of them produced fruits of very good shape and attractive appearance. The immature fruit color was medium green on the best of these selections.

The results obtained indicate that commercially desirable types of cucumbers may be isolated from crosses of either China or Puerto Rico No. 37 with good commercial varieties such as Colorado. Resistant plants have been obtained which approach Colorado in yielding ability, earliness, and fruit shape.

As far as could be determined, there was no linkage between the factors for resistance and those governing earliness or fruit shape. Possible linkage between factors for resistance and immature fruit color is being investigated.

Summary

Downy mildew is a common disease of cucumbers in many sections of the United States. It spreads by means of wind-borne spores and may be controlled by copper dusts or sprays. The development of downy mildew-resistant varieties would offer an easy and cheap method of control.

Out of more than 80 varieties and strains tested in 1939 only China and Puerto Rico No. 37 were highly resistant to downy mildew. These were crossed with Colorado, a good commercial variety, and the F_2 generations were grown in 1941.

The F_1 plants were intermediate for resistance. The F_3 is being grown for a genetic study of downy mildew resistance since an accurate classification of resistant types is difficult in the F_2.

Resistant plants of fairly good commercial type were isolated in the F_2.

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THREE GENERATIONS OF DEAF WHITE CATS

DOROTHY WOLFF*

DEFECTIVE hearing, according to Rawitz,[10] has been thought to be associated with albinism since 1769 when Buffon[5] speaking of albinos, said that in general those with weak eyes have weak ears. Buffon also referred to deaf white dogs.

In 1829 Bree[4] gave the first known report regarding deafness in the second generation of white cats. He had a deaf white Persian female cat which produced mixed litters but invariably the all-white offspring were deaf.

Blumenbach[3] in 1830 observed that the highly bred Persian cats are often deaf.

Sichel[13] writing in 1847 reported that for twenty years he had noticed a singular physiological observation which had been so constant each time as to be proven exact. "Cats which are entirely without a touch of color, and in which the iris is blue or blue-grey are constantly deaf."

According to this author, if the iris has a spot of yellow or a mixture of colored spots derived from the colors red or brown, the auditory functions are normal. Sichel's most interesting contribution to this subject is the report of a deaf white kitten with blue irides whose irides at the end of the fourth month became darker. At this same time the animal first commenced to show signs of attention when a ringing sound of sharp timbre was created, a little more than a meter from the cat.

The first attempt at post mortem examinations of these animals appears to have been reported in 1883, when Lawson Tait[15] described a profoundly deaf white cat which had occasional fits of epilepsy for four years. Postmortem examination revealed "triangular gaps" in the drum membrane which appeared to be congenital and were quite symmetrical. No examination was made of the inner ear. Tait stated that more male white cats are deaf than female.

The following year J. Stevens[14] reported a breed of deaf all-white cats with blue eyes, with one exception. One female had one blue eye, the other was pink. These cats were all females and exceptionally good mousers, for which reason they were bred. When offspring were "pied" or otherwise colored they were not deaf.

S. Sexton[12], referring to Lawson Tait's article stated—"abnormal hearing of this kind by no means establishes the fact that inner ear trouble does not exist since deaf mutes are very sensitive to grave or deep tones, thunder, for example, being even painful to them. Animals, like man, come to rely upon sound that can be 'felt' as it were."

Alexander Graham Bell[2] reviewing this subject in 1884 observed that statistics indicate deaf mutism is of less frequent occurrence in the negro race than in the white.

Darwin, under the subject of "Correlated Variability" in his *Animals and Plants Under Domestication*[6] stated that the organs of sight and hearing are liable to be abnormally affected in conjunction. He said—"kittens during the first nine days, whilst their eyes are closed, appear to be completely deaf." He believed that deafness in white cats with blue eyes is probably due to "a slight arrest of development in the nervous system in connection with the sense-organs."

The first complete autopsy of animals of this type with microscopic examination of the inner ear was made in 1896 by Rawitz[10] on a deaf white Dalmation dog. Rawitz found that the hearing center in the left temporal lobe on the right was only about half the normal size; that on the left was about one-third the nor-

*From the Oscar Johnson Institute, Washington University Medical School, St. Louis, Mo.

mal size. On both sides the fissure of Sylvius was extremely flattened. Examination of the cochlea showed high-grade changes, particularly on the left; on the right only two turns of the cochlea existed while on the left only one and a half. Spiral ganglion and acoustic nerve showed marked degeneration. Corti's organ, Reissner's membrane and stria vascularis were all destroyed.

The following year Rawitz[11] reported five animals, three dogs and two cats, with incomplete albinism and deafness. One cat had blue eyes and the other yellow. The blue-eyed cat appeared to be short-sighted. Both cats had dilated pupils. Microscopic examination showed advanced atrophy of both cochleae. Ossicles showed normal movement. Atrophy of the lowest gyrus of the temporal lobe of the brain was observed.

In 1900 G. Alexander recorded[1] a study of brain and labyrinth in an incompletely albinotic white cat and its embryo. The microscopic degeneration was similar to that observed by Rawitz. In addition Alexander noted degeneration of macula sacculi and the lumen of saccule was collapsed. He also observed the presence of pigment in an animal with one yellow iris. He saw no deviation in the brain from that of a normal cat. The embryo appeared normal.

In 1933 Howe and Guild[9] tested deaf white cats for sound transmission by the Wever-Bray technique. They got no changes of electrical potential.

In 1935 Howe[7] reported observations on six deaf ("albino"?) white cats. Uniform type of destruction of the organ of Corti occurred in all cases with one exception, one case being unilateral. In three ears there was only slight atrophy and disappearance of nerve fibers and cells in the spiral ganglion. Two of these ears were those of a kitten. Ganglion cell counts for normal and diseased cats were made. Counts for normal cats' ears ranged from 44,298 to 57,494 with a general average of 49,962. Eighteen albino (?) cats gave 23,854 to 55,052 with an average of 33,907. "In some cases a total destruction of the end-organ was accompanied by a normal ganglion cell count."

It is necessary to call to the reader's attention the fact that microscopic reconstruction of the spiral ganglion and its peripheral fibers reveals that an organ of Corti occurring in a single section cannot be correlated with the spiral ganglion cells of that section for, by the very nature of the organ, the peripheral fibers may spiral for some distance before arriving at their particular organ of Corti. The author has shown that only at the upper part of the basal turn (human) do fibers pass out directly to the organ of Corti, without spiralling.[17]

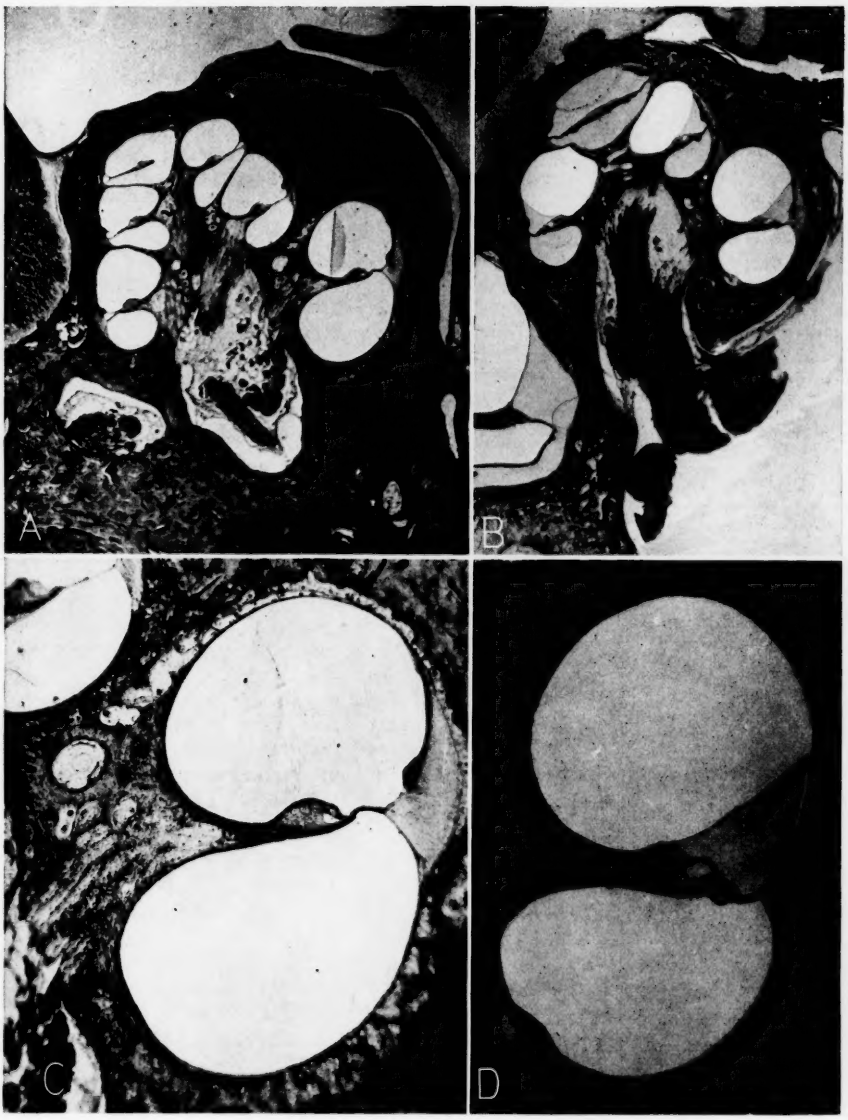
The following observations are presented with the hope that students of heredity may here find a fertile field of experimentation. Interesting crossings of deaf male and female cats might be made with a pure breed eventually developed. Observations as to the exact time of appearance of a fully developed organ of Corti in relation to the time when the kitten's eyes open, should be made; observations as to how soon the kitten loses its voice in relation to the degree of ganglion atrophy, etc., are also of interest.

Investigation of this sort on deaf white dogs had been planned but never executed by the late A. B. Duell.

Microscopic Observations on the Temporal Bones of Three Generations of Deaf White Cats and on One Other Sibling

The cats used in these experiments were bred by Dr. C. C. Bunch. The microscopic studies were made by D. Wolff.

The first deaf white female whose temporal bones were studied was mated with a normal white male cat. The deaf white female of this mating was then mated with its normal sire for the production of the third generation. The female progenitor was timid, never responded to any call and did not "meow" audibly. It was chloroformed after failure to deliver two of four kittens. In this particular batch of kittens no white ones had occurred. All four were predominantly black with some white spots. The deaf white female offspring to be described



SECTIONS OF EAR-ORGANS OF DEAF AND NORMAL CATS

Figure 3

A—is a mid-codiolar section through the cochlea of the deaf white female parent of two generations of deaf white cats. *B*—is a comparable section through the cochlea in a normal cat. The important landmarks are shown in the small drawing at the right. Note the absence of certain structures in the deaf ear which are present in the normal. *C*—is a detail of *A;* to be compared with *D* from the normal preparation. Note the absence of organ of Corti, and spiral ganglion cells. Reissner's membrane is in situ only in the apical and upper basal coils. It is not normal in these areas. The tectorium is distorted throughout.

later was born in an earlier litter.

Left Ear: Middle ear cavity, drum membrane and eustachian tube were normal. Tensor tympani muscle showed muscular atrophy with infiltration of fat. A great deal of impacted cerumen and cellular debris filled the external auditory canal.

No organ of Corti existed in any coil of the cochlea. No Reissner's membrane was seen in the basal turn but in the middle and upper turns it appeared agglutinated to the basilar membrane in such a manner as to reduce markedly the size of the cochlear duct. In the basal turn, inner sulcus cells were hypertrophied so that they practically filled the sulcus. Tectorium was also drawn into the inner sulcus and was scarcely recognizable. The cochlear aqueduct was quite wide open (as is true of normal cats) and was filled with delicate arealor tissue. Basal and middle turns of the cochlea showed marked atrophy of the spiral ganglion and nerve fibers. As few as eight ganglion cells were seen in one cut of the upper middle turn. A fair number of ganglion cells, 156, was present in the apex. Section 526 showed the spiral nerve sheaths, however, quite devoid of nerve tissue.

Vestibular end-organs and ganglion cells appeared to be in good condition. Perilymphatic vestibular spaces were distorted but this may have been an artefact of technique.

Right Ear: As on the left, middle ear was normal. Impacted cerumen and debris were in the external canal. Stapedius muscle was normal and tensor tympani on this side appeared to be in good condition although part of it was torn out as from poor infiltration.

An excellent mid-modiolar cut of the cochlea was secured (Figure 3*A*) so that one clearly saw in a single section the pathological condition from base to apex. As on the left, atrophy of ganglion cells was less in the extreme apex than in basal and middle turns. One cut of the upper middle turn showed only three ganglion cells present. In no turn was there an organ of Corti. Reissner's membrane was agglutinated to the basilar membrane in all turns. Tectorium was not recognizable in upper coils and in the basal, one could just discern where it should be. It appeared hyaline. No lumen was seen where vas spirale should be. See Figure 3*C* for detailed pathological changes.

Kitten (Term) F_1 (Not White) (Stillborn)

Right Ear: Middle ear was still well-filled with mesenchyme. Some mesenchyme persisted in scala tympani and scala media.

Tectorium was well-formed. The mound of the organ of Corti was of good size and pillar space was readily seen but pillar cells and hair cells could not be distinguished in the lower basal turn. An exceedingly large blood-vessel filled with blood lay immediately under the location of the organ of Corti in the upper basal coil. No lumen was seen in the location of the vas spirale; no tectorium, and no Reissner's membrane were visible. An abundant supply of nerve fibers appeared in Rosenthal's canal but spiral ganglion cells while present were not clearly stained, and scattered among these cells were large spaces filled with red blood cells. An aborted set of pillar cells but no real tunnel was noted. In the next higher coil a small tunnel space was seen, four nucleated cells on the lateral side of this and a nondescript mass medial to it. Tectorium was normal in this area. Reissner's membrane was present but not in situ. Only in the extreme apical turn was there an organ of Corti where three outer hair cells could be distinguished. The tunnel space was malformed and the inner hair cell area was hypertrophied.

Vestibular end-organs appeared to be in quite good condition. Mesenchyme was still present in perilymphatic spaces.

Left Ear: Three sections through the modiolus of the left cochlea were the only ones stained on this side. Essentially the same type of pathological change was observed as that on the right. The organ of Corti was imperfectly formed in all turns although the height of the mound was quite normal. This did not appear to be merely an embryonic state of development. The

vacuolated areas in the region of spiral ganglion cells were especially obvious in these cuts. As on the right, the supply of nerve fibers appeared abundant.

White Female Cat F_1
(Sibling of stillborn kitten described above)

This was also a deaf cat, very timid and the mother of two kittens, one of which was white, — the other "tiger" colored.

Left Ear: The similarity in the pathological picture here and that of the deaf mother was striking. No organ of Corti was present in any turn. Marked atrophy of the spiral ganglion cells was noted throughout, being more complete in the lower turns. But the atrophy in the apical region was much more marked than in that of the mother. Rosenthal's canal was in many areas devoid of fibers. No tectorium or Reissner's membrane was present. Both external and internal sulcus cells were hypertrophied.

Right Ear: A slight otitis was present in this ear, there being some evidence of cholesterin crystal imprints in niche of the oval window and a patchy infiltration in the submucosa.

As on the left, there was no organ of Corti throughout. The degree of loss of spiral ganglion cells and nerve fibers while marked was not quite so marked as on the left. Otherwise the picture was the same. Atrophy of the tensor tympani with fatty infiltration was seen here, as was noted in the left ear of the mother.

A mass of cerumen and desquamated epithelium was present in the external canal. This appeared to have broken through the drum in one area and was probably the source of the mild otitis media which was present.

F_2—White Kitten of the above Mother
(Granddaughter of the first deaf cat)

No organ of Corti and no Reissner's membrane or tectorium were present. In contrast to the parent and grandparent there was approximately a normal supply of spiral ganglion cells and nerve fibers. Middle ear and drum membrane were normal. Impacted debris was in the external canal. This kitten (third generation) was lively and apparently normal at first. It meowed as loudly as the male of the same litter. The animal was sacrificed when it was two months old. At this time it did not seem to respond to loud noises as promptly as the brother nor to meow as loudly. The kitten had blue eyes. None of the white cats in this series were true albinos. All had blue eyes.

Summary

Histological studies of the cases of three generations of deaf white cats are reported.

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APING THE MAN, OR MANNING THE APE

THE purpose of Professor Hooton's latest little book is to set forth certain facts which seem to prove that bodily form and modes of conduct are closely connected. He holds that this is true for both animals and man. Such a conclusion is most important eugenically as well as otherwise. If special types of physique are associated with special types of conduct and character, the eugenic world of the future will have another means of creating social taboos which will reduce the number of undesirable marriages. Part of the taboos of this kind which we have among us today are good, and part are bad. We have genuine, although not absolute taboos against marriages between rich and poor, Ph.D. and factory girl, Jew and Gentile, white and black, people across the railroad track. In a thoroughly eugenic world the main taboo will be against marriages and child-bearing among persons whose biological inheritance renders them unfit to produce and train children. There will be other taboos against marriages of the highly fit with those at a low level of fitness, and among people whose innate weaknesses are similar. The development of these desirable taboos would be facilitated if external bodily form could really be considered a reliable indication of innate capacities and tendencies.

Professor Hooton follows Cesare Lombroso, the Italian physician and anthropologist, in believing that bodily build and character are genuinely related. "The principal theme of all his (Lombroso's) theories are that crime is primarily a biological phenomenon; that the cause of crime is to be sought in the inferior organism of the individual. . . . I am inclined to think that, in spite of errors due to faulty thinking and primitive techniques, he alone can contest with Darwin the honor of being acclaimed the greatest anthropologist of them all." This is strong language, and many people will violently contest it. Nevertheless, Hooton's own exact study of more than 9,000 criminals, together with the facts set forth in the present book, warrant us in withholding judgment until the results of recent investigations, which Hooton regards as epoch-making, can be appraised.

Among these investigations Hooton gives high rank to those of George Draper and W. H. Sheldon. The latter's recent book (1940) is said to go far beyond anything previously published in classifying people according to bodily form and thus making it possible to compare people's physical attributes with their mental and cultural attributes. From body build alone, so we are told, Draper and his associates can predict whether a patient is suffering from gall bladder or gastric ulcer. They "can estimate with fair accuracy the environmental adaptability of a young adult male by appraising the degree to which he shows certain modernization characters in his physique." Knowledge of a person's physical characteristics enables Sheldon to frame sketches of personality and motivation which "are often marvellously close approximations to truth."

Hooton leads up to these final conclusions by examining the bodily form and mental reactions of lemurs, monkeys, and apes. Then he attempts a similar study of various species of man before *homo sapiens*. This is interesting, but not conclusive because so much of our knowledge of early man's habits, motives, and mental reaction is little more than guesswork. Coming down to living races, Hooton illustrates his method by means of a comparison between pygmy Negritos and full sized Negroes, "the tiny Negritos are always in the so-called refuge areas." They show a correlation between race, that is, bodily form, and culture "mainly in that the pygmies have almost no culture at all. . . . The signifi-

(*Continued on page* 80)

**Why Men Behave Like Apes and Vice Versa*, or *Body and Behavior*, by EARNEST A. HOOTON. 234 pp. Princeton University Press, 1940.

THE INCIDENCE AND MENDELIAN TRANSMISSION OF MID-DIGITAL HAIR IN MAN*

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HAIR is present on the basal segments of all fingers of the human hand, and is absent from the terminal segments. Individuals vary, however, in the presence or absence of hair on the mid-digital segments of the four fingers exclusive of the thumb. Both the presence and absence of mid-digital hair are so well distributed in white populations that the trait would have wide usefulness in chromosomal linkage studies, and in any other studies requiring "normal" heritable traits, if the mode of transmission could be definitely established.

Danforth,[3] in a study of mid-digital hair in 80 family groups, and in other groups classified by sex, racial origin, etc., concluded that "what seems to be a phylogenetically progressive loss of hair is brought about through the action of one or more recessive genes, or of one primary recessive gene with several modifying factors that regulate the distribution of hair when it is present" (p. 199). He also showed that the trait has anthropological significance.

In studies conducted by our laboratory primarily to search for linkage relationships in human traits, the distribution of mid-digital hair has been recorded routinely. A hand magnifying glass has been used to detect the presence of hair follicles on fingers with no visible hair, and in the present study, fingers showing follicles but not hair, as well as those bearing hair, have been counted as affected. No linkages have yet been established between mid-digital hair and other traits, but sufficient data are now at hand to permit further analyses of the incidence of mid-digital hair by age, sex, and natio-racial origin, its distribution among four digits of the hand, and its transmission in families.

Four sets of data are available:

1. *M.F.S. data:* 30 two-generation families located in a field study of myopia. At least one offspring per family had been a clinic patient at the Manhattan Eye, Ear, and Throat Hospital. The data comprise 201 subjects, 85 males and 116 females. Collected by Burks and a field assistant.
2. *H.H.S. siblings:* 99 pairs of two siblings and seven sets of three siblings studied at Huntington High School, Long Island. These include 100 boys and 119 girls of high school age. Collected by Burks.
3. *H.H.S. families:* 13 two-generation families — parents and siblings of H.H.S. pupils. These families were selected for follow-up because of large sibships. The data comprise 90 subjects, 45 males and 45 females. Collected by Burks.
4. *Supplementary families:* 8 two-generation families from among the acquaintance of the writers, included in order to gain further light on problems of diagnosis. The 30 subjects included 15 males and 15 females. Collected by Burks and Bernstein.

Although an effort was made to ascertain whether all offspring of a family group were full siblings, it is quite possible that concealed adoptions or illegitimacy may have influenced the results to some degree.

Influence of Age and Sex on Incidence of Mid-digital Hair

Danforth,[3] studying sections of skin of foetal and adult material concluded "that the (mid-digital) hair may be definitely present or absent from a very early period of life of the individual" (p. 191). We do not have sufficient data from very young subjects to check upon this finding, although it is interesting to note that the three youngest individuals among our subjects, aged 1, 1½, and 2½ years respectfully had no visible mid-

*The writers wish to thank Professor C. H. Danforth for a critical reading of the manuscript.

digital hair nor follicles, while two subjects of four years did have mid-digital hair. We have not included children younger than four in our subsequent calculations. It is quite possible, however, that hair might be visible on the fingers of young children and infants whose activity has not rubbed it off, and in fact one of us has observed it incidentally in an infant of 11 months.

With respect to sex incidence Danforth found that "in all cases of comparable groups from two sexes it is the female group that shows the higher percentage of cases without hair on the mid-digital region" (p. 196). In his group of several hundred American whites, 39 per cent of the males and 54 per cent of the females lacked mid-digital hair. Danforth suggested, however, that the difference may be due at least in part to the greater frequency in the female of very rudimentary hair which may not always be detected.

A sex comparison of our data revealed no significant difference in the percentage (69%) of female and (67%) of male students of the H.H.S. with mid-digital hair. The ages of the students range from 14 to 22 years, the great majority being 15 to 18 years of age. For the offspring generation (ages 4 to 39) of the M.F.S. and H.H.S. family groups, no significant sex difference was found in age groups under 18 years of age (see Table I). Moreover the incidence of mid-digital hair showed no marked age trend in the males, but dropped decidedly in the group of females over 21 years of age.

The data strongly suggest that Danforth's explanation of sex differences is a sound one, but that further, the difficulty in the diagnosis of the mid-digital hair condition in females is not obtrusive before maturity, especially in environments where the demands for manual work are not heavy. After maturity, however, diagnosis of either mid-digital hair or follicles becomes uncertain in women (and probably also in men, but to far less degree). In our group of supplementary families collected with special reference to factors influencing the diagnosis of mature females, it was found that several women who appeared to be asymmetrical at first examination proved to have follicles that would balance the symmetry when re-examined with a 12-diameter lens. There were also cases with a few pits in the skin which we could not be sure were follicles.

Natio-racial Variation

Danforth reported that "the Indian, the Negro, and the Japanese, as races, have gone farther toward freeing the mid-digital region of hair than has the white race, although certain members of the latter race have reached a stage quite as advanced as that of any representative of the colored races" (p. 197).

In our data, although histories of natio-racial descent were not secured, it was possible to make some rough appraisals of the trait incidence in several groups by classifying subjects according to family name and hair color. This was done with the H.H.S. students, for whom the conditions of data collection were more uniform than with the M.F.S. group. Subjects were used in this treatment only if the family name belonged almost unmistakably to one or another nationality. A small number of Negro pupils were also considered as a separate group.

We believe (from general knowledge and observation) that the Italian and Irish groups in this country have undergone relatively less natio-racial mixture than many other groups. We have accordingly classified them separately, and have grouped the other families so as to yield the classes tabulated in Table II.

The subjects of Irish derivation appear to have less mid-digital hair than do other North Europeans, and the Italian subjects have even less, especially if they are dark-haired. Our limited data on Negroes are in accord with those of Danforth in showing little mid-digital hair. Although a classification based only upon family names and hair color admittedly lacks rigor, the expected effect of inaccuracies in grouping would be to obscure rather than accentuate natio-racial differences. The differences which do emerge as statistically significant, therefore, are probably real ones, and suggest the desirability of further study.

Combination Patterns and Symmetry

Our H.H.S. cases were examined for combination of fingers bearing mid-digital hair. For simplicity only the left hands were tabulated.

In 140 of 151 affected cases, i.e. in 92.7 per cent, the combinations reported by Danforth as most frequent were found to hold. The distribution of all cases showed:

No. of affected fingers	Fingers affected	Percentage Incidence in 219 cases
0		31.0
1	4th	16.0
2	3rd, 4th	14.2
3	3rd, 4th, 5th	28.8
4	2nd, 3rd, 4th, 5th	5.0
	other combinations	5.0

In six of the 11 cases in which the left hand bore mid-digital hair in an atypical combination, the right hand was in accord with the "combination rule." In the 16 cases in which one or both hands bore mid-digital hair contrary to the combination rule, 13 were asymmetrical with respect to right and left hand.

Danforth states that in about nine out of ten cases the two hands of one person were found to be affected similarly with mid-digital hair (p. 194). Our H.H.S. indices of symmetry were found to be 92 per cent for presence or absence of hair (or follicles); but the percentage of these cases in which the two hands were found to be affected in exactly the same combination was lower, i.e. 74 per cent. Out of 56 subjects showing asymmetry, 13 or 23 per cent had at least one hand in which the mid-digital hair distribution deviated from the combination rule.

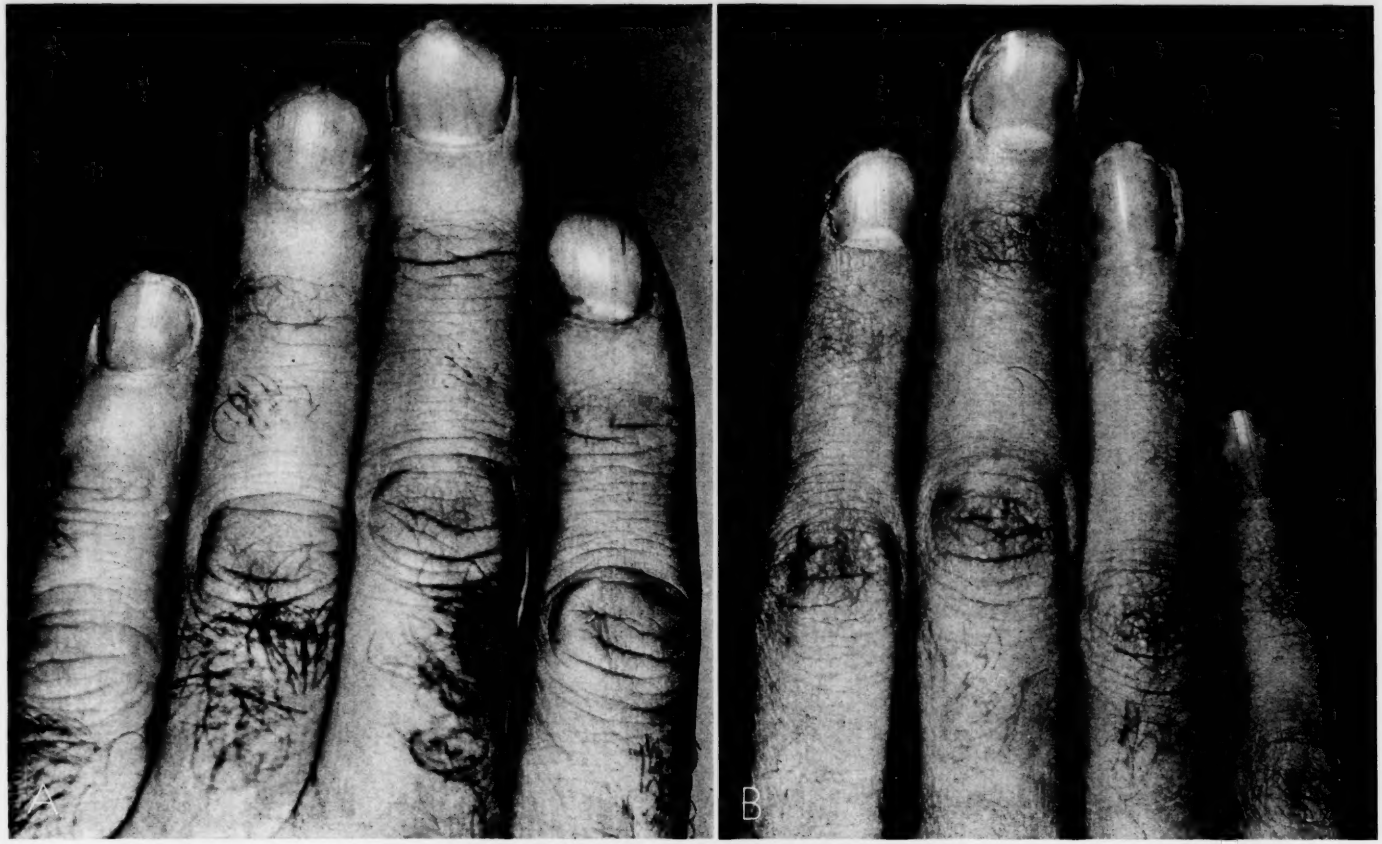
It is of course possible, (and even strongly suggested by our age data), that cases departing from the combination rule or from symmetry are mainly those from which hair and evidence of follicles have disappeared from certain fingers.

Tests for Monomeric Inheritance and Dominance

Family data. The M.F.S., H.H.S. and Supplementary family data provide 36 family groups that can be used for analying F_1 ratios for various parental combinations. The results are shown in Table III.

The incidence, q, of the allele (or combined alleles) for the condition assumed to be recessive has been estimated by calculating the square root of the proportion of non-affected individuals (in the total family population of parents and offspring combined). Cases asymmetrical in the two hands with respect to affected and non-affected conditions and cases contrary to the combination rule have not been used in the treatment which follows. Since there is not a sufficient number of matings representing all the combinations of affected and non-affected fingers, it has been necessary to make certain phenotypic groupings, viz. any fingers affected *vs.* none affected; 4, 3, or 2 fingers affected *vs.* 1 or none affected; 4 or 3 fingers affected *vs.* 2, 1, or none affected. If Danforth's conclusion that "the presence of hair in almost any degree is generally dominant over all conditions representing less amounts" (p. 198) is valid, such groupings should provide a sound test of monomeric inheritance even if two or more alleles should be lumped together in classifying affected and non-affected individuals. Such data might, on the other hand, provide evidence with respect to Danforth's alternative hypotheses accounting for the phenomenon "through the action of one or more recessive genes, or of one primary gene with several modifying factors that regulate the distribution of hair when it is present" (p. 199).

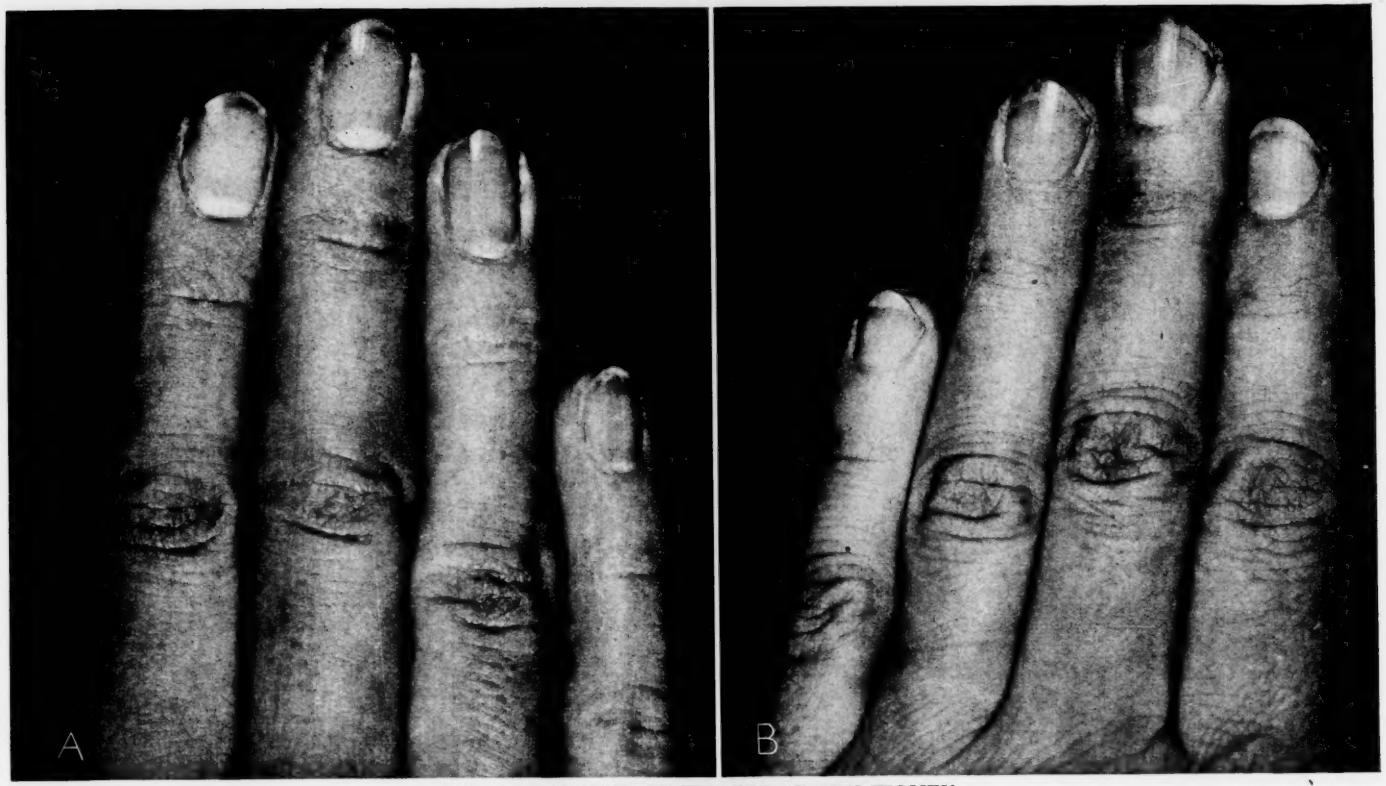
Inspection of Table III would at first suggest that a monomeric hypothesis is untenable, since matings between two "non-affected" parents produce an appreciable number of affected offspring, especially when no finger or one finger affected are presumed to be the recessive conditions. It is relevant to recall, however, the age-decrease in the incidence of mid-digital hair which we found in women over 22 years of age. This would mean that some of the mothers recorded as non-affected are probably affected genotypes. By way of interest we calculated the probable number of genotypically affected mothers included in the "non-affected" matings, on the assumption that the true proportion of affected mothers is the same as the observed



MID-DIGITAL HAIR ON FINGERS OF TWO MEN

Figure 4

A—fingers 3, 4 and 5 carry mid-digital hair; *B*—mid-digits of fingers 3 and 4 are sparsely haired. All of the basal joints carry hair or hair follicles. See Figure 5. (The authors wish to acknowledge with appreciation the cooperation of the Department of Zoology, Columbia University, and of Mr. Jack Godwich, photographer for the Department who provided the photographs.)



MID-DIGITAL HAIR ON FINGERS OF TWO WOMEN

Figure 5

A—fingers 3, 4 and 5, show mid-digital hair follicles but the hair has been rubbed off; *B*—all fingers show absence of mid-digital hair or of hair follicles. These four figures show several patterns of distribution of hair on the fingers of four individuals. All four fingers of the two men show hair on the basal segments. The two females show only hair follicles on these segments in the photographs, although rudimentary hair could be detected in examination of the actual fingers. The hair is lost earlier on the fingers of women than of men, but the follicles remain.

proportion of affected fathers. The number of such cases (entered in Table III) is clearly just about sufficient to account for the affected offspring appearing contrary to Mendelian expectation.

In the matings of two affected parents, where there is no question of affected mothers failing to show up, only the presence *vs.* absence of mid-digital hair comparison provides sufficient cases for calculating expected affected and non-affected offspring. The agreement between expectation and observation is seen to be high.

In the matings which include one affected parent the observed and expected offspring ratios show fairly close agreement. As with "both parents non-affected," the presence of a few genotypically affected mothers who are classified as non-affected would tend toward an excess of affected offspring, but a slight error in the other direction due to omitting asymmetrical offspring has more weight in these matings than in the other matings. The net result is a slight excess of non-affected cases.

Sibling data. In order to utilize the larger series of H.H.S. data which included sibling pairs, but not their par-

TABLE I. Effect of Age and Sex on Incidence of Mid-digital Hair (Offspring generation of M.F.S. and H.H.S. families, right hand)

Age	No.	Proportion of indiv. affected	Standard error of prop.	Average no. affected fingers per indiv.
Under 14				
Males	25	.56	.10	.96
Females	24	.50	.10	.96
14-17				
Males	25	.52	.10	.88
Females	41	.51	.08	1.07
Under 18 (total)				
Males	50	.54	.07	
Females	65	.51	.06	
18-21				
Males	28	.57	.09	1.11
Females	23	.48	.10	.96
Over 21 (to 39)				
Males	21	.48	.11	1.00
Females	31	.19	.07	.36

TABLE II. Mid-digital Hair by Natio-racial Groups and Hair Color (H.H.S. data)

Group	No. of individuals	Proportion of indiv. affected	Standard error of prop.	Av. no. of affected fingers (Left hand)
North European				
All cases	111	.78	.04	1.97
Cases with light hair or having light-haired sib.	63	.79	.05	2.08
Irish sub-group*	38	.55	.08	1.37
Italian				
All cases	58	.55	.06	1.36
Cases with dark hair and having dark-haired sib.	37	.43	.08	1.16
Negro	8	.12		.03**
Hair color (matched to standard chart)***				
Blond	27	.78	.08	2.07
Light medium	41	.76	.07	1.68
Dark medium	57	.74	.06	1.86
Dark	83	.64	.06	1.34

* H.F.S. offspring generation cases were included in order to secure more cases for comparison.

** Only one of the eight Negro pupils had mid-digital hair, and on only the 4th fingers.

*** Only white subjects were used for this comparison.

TABLE III. Offspring Phenotypes in Mid-digital Hair for Three Types of Matings (H.H.S., H.F.S., Suppl. families.

"Affected" have hair on fingers:	PARENTAL COMBINATIONS: Both P affected — Offspring: Aff.	Both P affected — Offspring: Non-aff. Omitted	One P affected — Offspring: Aff.	One P affected — Offspring: Non-aff. Obs. Exp. Omitted	Both P non-affected — Offspring: Aff.	Both P non-affected — Offspring: Non-aff. Omitted	Estimated combined incidence of genes responsible for mid. affected phenotype.
2,3,4,5 * or 3,4,5	4	— — (2 families)	15	17 15.3 4 (9 families)	3	109 1 (25 families) 1 mother may be aff.	$A_2 A_1{}^A{}_0$ .917
2,3,4,5 ** 3,4,5 or 3,4	11	4 — (4 families)	17	21 17.2 7 (12 families)	10	58 4 (16 families) 3-4 mothers may be affected.	$A_1{}^A{}_0$ .829
2,3,4,5 *** 3,4,5 3,4 or 4 only	45	8 9.0 3 Obs. Exp. (12 families)	22	17 16.1 7 (12 families)	7	47 1 (12 families) 3 mothers may be aff.	A_0 .704

* Affected mothers are 3 per cent fewer than affected fathers.

** 23 per cent fewer affected mothers. There are four families included in the other phenotype comparisons which could not be here included because of parents failing to meet our "symmetry" criterion with respect to affected and non-affected conditions.

*** 84 per cent fewer affected mothers.

TABLE IV. Tests of Monomeric Inheritance and Dominance of Mid-digital Hair over Absence or Lesser Degrees of Mid-digital Hair. (H. H. S. siblings.)

Phenotypic contrast	No. of subjects	Estimated q	Affected sibs of sibling pairs	Pairs observed	Pairs expected	Random no. pairs expected	Monomeric hypoth. chi-square	Monomeric hypoth. P (approx.) (for n=1)	Random distribution hypoth. chi-square	Random distribution hypoth. P (for n=2)
I D: $A_4 A_3 A_2 A_1$	127,	.5196	0	14	13.6	6.2				
	*		1	19	19.8	34.3	.047	.8	17.4	<.01
R: A_0	47		2	54	53.6	46.4				
15 sibships omitted										
II D: $A_4 A_3 A_2$	84	.7071	0	29	30.6	21.0				
			1	26	22.8	42.0	.6165	.5	12.1	<.01
R: $A_1 A_0$	84		2	29	30.6	21.0				
18 sibships omitted										
III D: $A_4 A_3 A_2$	112	.6633	0	29	30.4	19.4				
			1	30	27.1	49.3	.4210	.6	15.3	<.01
R: $A_1 A_0$	88		2	41	42.4	31.3				
Atypical cases added (16 prs.)										
2 sibships omitted										
IV D: $A_4 A_3$	51	.8124	0	39	40.6	32.7				
			1	21	17.7	33.6	.8515	.4	10.5	<.01
R: $A_2 A_1 A_0$	99		2	15	16.7	8.7				
27 sibships omitted										
V D: $A_4 A_3$	86	.7461	0	39	41.1	30.0				
			1	30	25.8	48.0	.9513	.3	13.8	<.01
R: $A_2 A_1 A_0$	108		2	28	30.2	19.0				
Atypical cases added (22 prs.)										
5 sibships omitted										

ents, a method presented by Cotterman was applied. When data comprising only a single generation are available, it is possible to test for inheritance through a single dominant gene substitution by a comparison of observed and expected frequencies of sibling pair combinations. In the present instance the use of this method has the distinct advantage that the older subjects (i.e. the parents and particularly the mothers), some of whose phenotypes have changed with age, can be disregarded.

For the case of two sibs per family, the expected percentages of sibling phenotypic combinations are:

Both dominant $\frac{1}{4}\ p\ (4 + 5p - 6p^2 + p^3)$
One dominant $\frac{1}{2}\ p\ (1\text{-}p)^2\ (4\text{-}p)$
Both recessive $\frac{1}{4}\ (1\text{-}p)^2\ (2\text{-}p)^2$

where p is the incidence of the dominant gene.

Only white families were used, and only two siblings per family. In the families having more than two children examined, the two older children were arbitrarily selected for this comparison. Cases were omitted when the combinations of fingers bearing mid-digital hair were contrary to the "combination rule," or when one hand showed the affected and the other hand the non-affected phenotype. The numbers of siblings thus omitted are shown in Table IV.

There is marked agreement between observed and expected frequencies of sibling pair combinations, the chi-square test showing the deviations to be within ordinary chance range.*

As in the previous test based on parents and offspring, certain phenotypic groupings were necessary to insure an adequate number of sibling pairs. Thus in the first comparison, the presence of hair on 1, 2, 3, or 4 fingers was assumed to be dominant (D) over recessive absence of hair (R). In the second comparison the presence of hair on 2, 3, or 4 fingers was assumed to be dominant over absence of hair on only one finger. In the fourth comparison, hair on 3 or 4 fingers was assumed to be dominant over 0, 1, or 2 affected fingers.

In the third and fifth comparisons cases previously excluded on the ground of asymmetry or failure to agree with the combination rule were added and classified with the four or three affected fingers group if at least one hand had finger 5 affected (with hair; not with follicles alone); with the two or more affected fingers group if at least one hand had hair on fingers 3 or 5 or both. Our preliminary study of affected finger combinations had shown that if finger 5 was affected, fingers 3 and 4, or 2, 3 and 4 were in general affected also. It therefore seemed legitimate to test the assumption that certain atypical cases were genetically equivalent to typical cases. It is seen that when sibling pairs containing atypical cases as defined are added the agreement between observation and expectation is about as good as when these pairs are not used. Further investigation would be necessary, however, before we could equate the atypical cases to the typical ones with any assurance.

Returning, now, to the hypotheses which have been considered to account for mid-digital hair, we believe the evidence favors monomeric rather than polymeric inheritance, and that the dominance of hair in almost any degree over lesser amounts occurs too regularly to

*It may be noted however, that the deviations tend to be in the same direction for most of the corresponding comparisons, e.g. that the expected frequency of pairs with one affected and one non-affected member tends to be lower than the observed. It may be supposed that this is a consequence of estimating q (and $p = 1\text{-}q$) from the proportion of non-affected individuals, some of whom may actually be affected genotypes. Errors in diagnosing the affected condition would result in underestimating p, which in turn would have more influence upon the calculated expectation for pairs with one affected member than upon the other calculated expectations, a fact evident from the formulae. The question may be raised as to why p estimated from chi-square is given on the basis of only one degree of freedom when there are three pairs of entries corresponding to each estimate. Cotterman, p. 129, explains the loss of two degrees of freedom in accordance with the fact that the expected frequencies have been adjusted to agree with the observed series in two respects, i.e. their total number and the proportion of recessives from which the gene frequency is estimated.

be compatible with a hypotheses of a single main gene substitution with modifiers regulating distribution.

A Multiple Allele Hypothesis

One of us (Bernstein) has envisaged a hypothetical series of alleles A_1, A_2, A_3, A_4 (in order of increasing dominance) with subscripts corresponding to the number of fingers affected with mid-digital hair, and A_0 in homozygous condition corresponding to absence of mid-digital hair. We do not have sufficient data to make separate tests for the presence of each of these alleles and in particular our data do not permit a satisfactory test to distinguish between A_3, A_4. The A_3 and A_4 conditions are clearly differentiated, however, from the other conditions, both in the family data classified by matings, and in the H.H.S. sibling data. It is possible that A_3 and A_4 are sometimes genetically equivalent and sometimes overlapping, since Danforth not only reports two A_4 siblings whose parents are both A_3 but also reports far more offspring who are not A_4, though having an A_4 parent, than would be compatible with a separate A_4 allele. In our own data we also have two A_4 siblings whose parents are both A_3, which raises the question of A_4 sometimes representing a homozygous condition of A_3. But we also have three families with A_4 offspring in which the father is A_4, and one family with an A_4 child in which the mother is A_3, the father A_1, which again raises the question of inadequate diagnosis of older females.

As a further check we took all A_4 subjects of offspring generation from the family data and H.H.S. siblings, and computed the proportion of A_4 cases among their siblings. Each family was used as often as it contained A_4 cases, so that the expected proportions of affected siblings assuming A_4 to be a rare dominant, would be 50 per cent. The result of this examination gave 18 A_4 offspring whose 29 siblings included 10 or 34 per cent A_4 cases, and 11 or 38 per cent A_3 cases.

Aside from the question of an A_3, A_4 distinction, our H.H.S. sibling data are consistent with a hypothetical A_4 or A_3, A_2, A_1, A_0, and our family data are consistent if appropriate allowance is made for genotypically affected mothers occurring as non-affected phenotypes.

Summary and Conclusions

In a study of the distribution of mid-digital hair in man with especial reference to problems opened by Danforth's previous study:

1. No significant difference in sex incidence was found in subjects under 18 years of age. The males showed no marked age trend after this age, but the females showed a decided drop in affected cases after age 21. This result is interpreted both in terms of the higher incidence in females of very rudimentary hair and their more frequent performance of manual work (housework), which may wear away mid-digital hair and obliterate the follicles.

2. Subjects of Irish derivation appear to have less mid-digital hair than do other North Europeans, and Italians have even less, especially if they are dark-haired. Our limited data on Negroes are in accord with those of Danforth in showing little mid-digital hair.

3. The combinations of 1, 2, 3, and 4 affected fingers reported by Danforth as most frequent were found to hold with few exceptions in the present data. The proportion of our cases (of high school age) whose two hands were affected symmetrically was 74 per cent, as compared with about 90 per cent in Danforth's groups. There is reason for believing that hands departing from the "combination rule" are phenotypic deviates, since in nearly all such cases the right and left hand are asymmetrical with respect to each other.

4. Danforth's finding that with very few exceptions a child will have mid-digital hair on no more fingers than its more hairy parent was confirmed. One of the authors (Bernstein) offers a multiple allelomorph hypothesis, A_0, A_1, A_2, A_3, A_4 to account for distinction of the hair on the digits. If A_3 and A_4 are in fact separate alleles, there appears to be phenotypic overlapping. Aside from this distinction, our two generation pedigrees are consistent with the multiple allele

hypothesis if appropriate allowance is made for genotypically affected mothers occurring as non-affected phenotypes. Again ignoring the A_3, A_4 distinction, our single generation results on siblings of high school age, examined by Cotterman's method of testing for monomeric inheritance with dominance, are even more consistent with the multiple allele hypothesis.

5. In view of the excellent agreement with Mendelian theory obtained from siblings showing regularity in the distribution of mid-digital hair, but the difficulty experienced in diagnosing certain cases, particularly among mature women and among infants, it would be desirable in order to facilitate use of this trait in human genetics work to pursue further research upon the conditions that permit rigorous classification of the pilosity of subjects.

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A CASE OF VIABLE TWIN CHICKS

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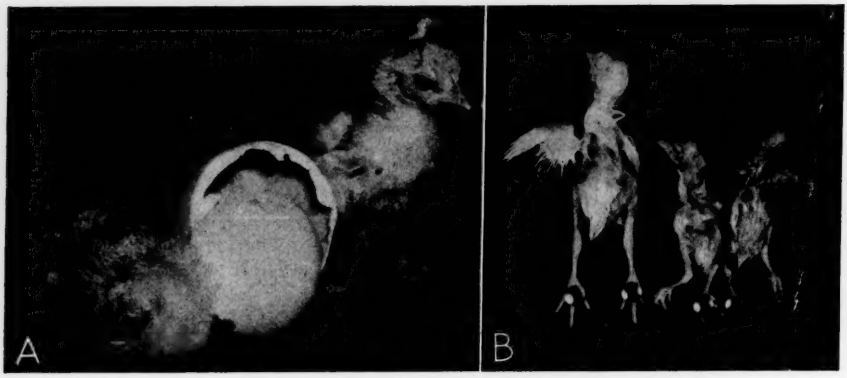
THE recent reviews of twinning in this JOURNAL by Newman[1] and Waddington[2] make it seem desirable to report a case of twin chicks which lived for a short time after hatching. The case seems to be of sufficient rarity to merit a brief description.

The twins (of the Austra-White breed) were hatched on July 15, 1941, at Pettys' Hatchery near Urbana, Illinois, and the following information was given to the author by Mr. Pettys a few days after the event had occurred. During the routine removal of newly-hatched chicks from incubator trays, one chick (referred to as No. 2 below) was noticed to have almost emerged from an egg. With some help this chick freed itself from the shell, and a second chick (No. 1) was noticed on the bottom of the egg shell. According to Mr. Pettys there is no doubt that both chicks emerged from the same shell. No. 2 seemed healthy and capable of moving around freely, in spite of the fact that the toes on the left foot were crooked (Figure 6*A*). Both chicks were fully covered with down and No. 2 showed fairly well-developed primary wing feathers. No. 1 could not stand due to the fact that the right leg was "deformed." This chick died when it was 24 hours old and I did not see it alive, while No. 2 was still alive two days after hatching. It appeared normal in all respects except size (Figure 6*B*). It died when it was two days old. Both dead chicks were made available to me for inspection soon after the second chick died. Even though the dead chicks had been kept in the refrigerator they were in the beginning stages of decomposition. The chicks were weighed and autopsied. No. 1 weighed 11 grams. No. 2 weighed 16 grams. Because the weight of the chick is positively correlated with the weight of the egg, one would expect normal chicks of the Austra-White breed to weigh from 35 to 45 grams at hatching. The viscera appeared normal by inspection but could not be removed from the birds without tearing because of beginning decomposition and no comparative



TWIN CHICKS

Figure 6

A—The twins a few hours old (photograph by Ben Parker, Urbana). *B*—Comparison in size of the twins with a day-old White Leghorn Chick.

measurements could be obtained. The small size of the viscera was quite striking. The yolk sac of No. 2 had broken and no yolk sac could be found in No. 1. No. 2 had by far the greater amount of yolk (about 5 cc) in its abdominal cavity, while No. 1 had not more than a few drops of a thin, greenish substance which was assumed to be yolk. Both birds were of the female sex.

These chicks could have arisen from a single blastoderm, from two blastoderms on one yolk fertilized by two sperm (Newman[1]) or from an egg containing two yolks. While it is impossible to determine with certainty how the twinning occurred in this case the following points are of interest:

1. Mr. Pettys was asked whether it could have been possible that the chicks had emerged from a double-yolked egg. Following the custom of hatcherymen in general he would not have set a double-yolked egg (usually recognizable by its much larger size) which, from experience, would not have hatched. If one assumes that in this case the egg in question had been small and double yolked, it would probably have been noticed on candling the eggs at the time of the routine removal of the infertile eggs and eggs containing "early dead" embryos.
2. The small size of the chicks (11 and 16 grams) may be considered as evidence in favor of their origin from a single-yolked egg.
3. Both chicks were females.
4. Presence of a yolk sac with yolk in one chicken and its absence in the other is well in line with embryological expectations in a case of this nature.
5. Careful inspection did not reveal anv differences in color of the down feathering.

The last three points support the possibility that these twins are of monozygotic origin.

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ADVENTITIOUS ROOTS ON PANICLES OF RICE

JENKIN W. JONES AND MERRITT N. POPE*

THE rice varieties grown commercially in the United States belong to the species *Orzya sativa* L. The fibrous roots originate at the crown of the plant and are most numerous near the surface of the soil. On submerged land adventitious roots often develop at nodes of the culm that are well above the surface of the soil and often above the surface of the water. Juliano and Aldama† in discussing growth of adventitious roots report that: "Development of root primordia takes place at every node and in the intercalary meristem on the stem of this variety (Inapostol) of rice under study and these only become apparently inconspicuous at the third node below the flag." The culms of the rice plants are hollow with solid nodes.

In August, 1939, when at the Rice Experiment Station, Crowley, La., N. E. Jodon‡ called the senior writer's attention to a few panicles of rice, of hybrid origin, on which small root-like structures, apparently originating in the region of the outer glumes, had developed at the base of some of the seeds.

In early September of the same year, panicles with similar outgrowth also were observed at Texas Substation No. 4, Beaumont, Texas, in a partly sterile strain of Caloro, selected from plants grown from X-rayed seed. Late in September at the Rice Branch Experiment Station, Stuttgart, Ark., a panicle of the Nira variety was collected on which the spikelets were nearly all inclosed by a canopy of relatively thick outgrowths many of which were longer than four inches (Figure 7*A*). Some of these were sectioned and proved to have the characteristic structure of roots (Figure 8*B-C*).

Since the stem of the panicle (Figure 7*A*) in rice is compressed lengthwise the individual nodal tissue is difficult to identify. The adventitious roots observed arise in this nodal tissue and emerge somewhere between the lemma and the abscission layer of the seed. Sections made of two seed bases show roots emerging above and below the second glume. Other seeds examined showed roots arising below the first glume also. In Figure 8*A*, the lemma and palea have been removed from the caryopsis of the center spikelet and the ungerminated embryo is seen. In all three spikelets the origin of the roots in relation to the glumes can be seen.

Inheritance

In the spring of 1940 normal Nira seed and seed from the panicle with roots were sown in pots. The seed from the latter was immature and only one plant survived, producing normal panicles. Plants from seed of the partly sterile strain of Caloro, however, produced roots on some panicles in 1939, and when crossed with normal Caloro segregated in a ratio of three normal plants to one partly sterile, some of which produced small roots on the panicles.

The development of root primordia may occur at each node on the culm of rice, but according to Juliano and Aldama†, they are inconspicuous in the Inapostol variety at the third node below the flag leaf. In the panicles of Nira and the partly sterile strain of Caloro, roots developed in the nodal tissue at the base of the seeds, which are located well above the flag leaf. Since certain growth hormones are essential for the development of root primordia and

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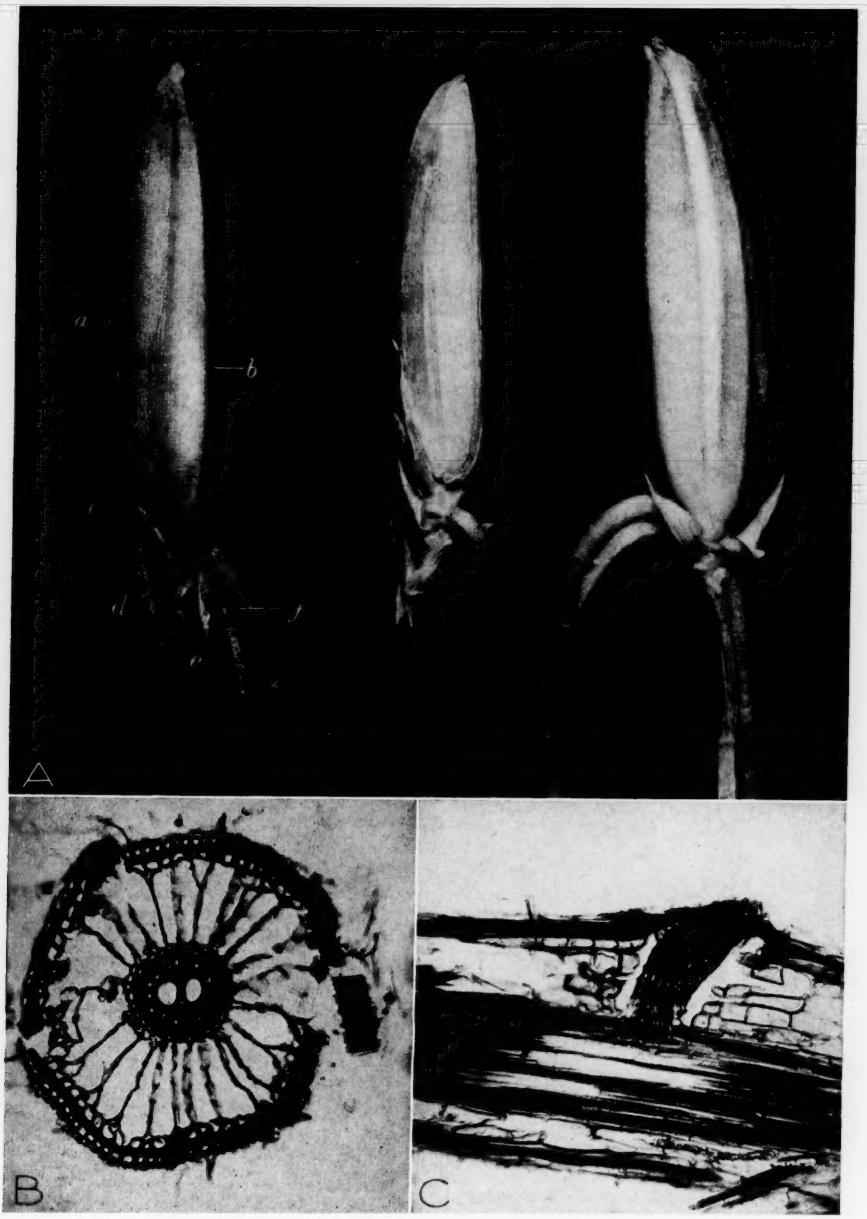
†Juliano, José B., and Marcos J. Aldama. Morphology of *Oryza sativa* Linnaeus. *Philippine Agr.* 26:1-134. 1937. (Page 14.)

‡Assistant Agronomist, Division of Cereal Crops and Diseases, Bureau of Plant Industry, United States Department of Agriculture.

STRUCTURE OF PANICLES

Figure 7

A—Normal panicle (right) and abnormal panicle (left) with canopy of roots. *B*—Sections of abnormal panicle showing seeds and roots.



STRUCTURE OF SEEDS AND ROOTS

Figure 8

A—Showing the point of origin of the roots on the seed. (*a*) Lemma, (*b*) palea, (*c*) glume, (*d*) abscission layer, (*e*) rachis, and (*f*) adventitious roots. *B-C*—Sections showing characteristic structure of roots from the abnormal panicle.

others for root elongation, such factors must have been present in the proper proportions and sequence in the panicles on which roots developed.

✦

METABOLIC TERMINOLOGY

ACCORDING to current usage the word "metabolism" has come to cover not only the combustion of fats, proteins, and carbohydrates, but also all chemical processes concerned in the exchange of substance between the organism or part thereof and its environment.

It now stands for every real and imaginable chemical reaction the organism is capable of, from those involved in the respiratory quotient, through those concerned in estrogen production, to those associated with bone formation. Genes, conceived of as being chemical agents directing the most fundamental of growth-processes, are even cited as taking part in the metabolic game, if only symbolically, in the present state of experimental knowledge.

The care-free abandon with which the word is used by workers in the biological sciences from biochemists to geneticists has endowed it with an intention it is etymologically incapable of carrying, and made it a waste-basket for vague and loose thinking. Perhaps such waste-baskets are necessary, but it may not be too much to ask that such should at least be able to hold that which is dumped in them.

The root *meta* means "away," "after," "beyond," and "between," and the root *bol* means "put" and "throw." By no stretch of the imagination can any combination of meanings carried by these roots be brought to stand for the chemical change and exchange of substance involved in the exercise of living.

Since in last analysis the concern of that which is called metabolism is nothing more nor less than substance utilization for energy production and expenditure, the roots of the term used for this general activity of living should approximate in meaning the basic attributes the word is intended to stand for, viz., substance and energy.

The word *plasma* meaning the "thing," "stuff," or "substance moulded"; and the word *ergon* meaning "work" and "energy," provide such roots. *Plasma* is chosen because of its restrictive association with living things; *ergon* because it may be taken to represent all manifestations of energy, be they chemical, thermal, electrical, or mechanical.

When the two are joined to form *plasergy* a word is had whose roots carry the meaning of work done in and by the substance of living. This can be extended without strain to stand for the chemical reactions involved in energy production, utilization, and expenditure in and by living organisms; and conversely for the energy exchange involved in cytoplasmic activity as derived from chemical change and exchange in substance.

Without difficulty *Anaplasergy* will cover the substance increase of living; and *Cataplasergy* its break-down; and so on similarly for other useful terms.

This coinage of a new word to replace one long in use will undoubtedly meet with vivid if not dismayed opposition. The only objections which can legitimately be voiced, however, are that the word is new, that it upsets custom, and that it lessens the use of a phonetically agreeable combination of syllables.

Against these emotional reactions stands the cold fact that in *plasergy* word symbol and subject come together, which is more than can be said for *metabolism*; and the fact that *plasergy* like other terms of general intent, such as growth, reproduction, respiration, and the like, stands for the product as well as the processes of activity. The word means something and sounds like what it means. With it there can properly be a *Journal of Plasergic* (Substance Utilization) *Research*, instead of one with the faintly truthful title of *Journal of Throw Away* (Metabolic) *Research*.

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THE INHERITANCE OF A HEMOPHILIA-LIKE CONDITION IN SWINE*

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HEMOPHILIA and blood conditions which resemble this abnormality have been observed in man for many years (Otto,[14] Nasse[13] and many others in recent years). A similar condition in swine has not heretofore been reported. Hemorrhages in other animals have been observed, but no genetic or blood chemical studies were made to determine if these hemorrhages were due to hemophilia. It is not likely that hemophilia would be as frequently observed in farm animals as in man, even though present, because fewer observations have been made on the blood of farm livestock. The presence of such a blood condition in animals should offer opportunities for research in physiology and therapy not possible with man because of the danger to the life of the experimental subject. The occurrence and mode of inheritance of an abnormal blood condition in swine which is similar to hemophilia in man is discussed in this article.

Literature

Fordyce[5] pointed out the hereditary nature of an abnormal blood condition in man which was called "bleeder" by Otto[14] and later named "hemophilia" by Schonlein.[16] Nasse[13] over a century ago stated that the abnormality was transmitted from normal (carrier) females to their sons. The daughters of hemophilic males were carriers whereas the sons were normal.

Most authors (Lukowski,[10] Birch[2] and others) observed hemophilia in the male only; but Warde,[19] Steiner,[17] Lloyd[9] and others have reported afflicted females. Either hemophilic females are less viable[1] or two families having the abnormality fail to intermarry[3] thus accounting for the very few cases of hemophilia observed in women. It is generally agreed that hemophilia in man is due to a sex-linked recessive gene.

Hemophilia in man shows considerable variation in the age of the individual when the character is first expressed (Mills[11]), but hemorrhages are rarely observed before the child is two years of age. Mills[11] also reported an increase in severity of the abnormality with age until puberty, after which there is generally less difficulty with hemorrhages.

Hemophilia is differentiated by Duke[4] from *purpura hemorrhagica* by a lower platelet count in the latter. However it has been shown that the platelets in hemophilia are abnormally stable.[8,12] Purpura hemorrhagica is also characterized by a non-retracting blood clot whereas in hemophilia the blood clot retracts in a normal manner.[15,18]

A hemophilia-like diathesis characterized by a prolonged bleeding time and a normal coagulation time which occurs in both sexes has been called pseudo-hemophilia.[6]

Materials and Methods

A hemophilia-like condition was observed in some foundation animals of one strain of Poland China swine used in the Regional Swine Breeding Laboratory project at the Missouri station. When inbreeding was practiced, the incidence of the abnormality became greater until a selection program against the condition was employed. The swine used in the inheritance studies consisted of animals in the swine breeding project and cross-

*Contribution from the departments of Animal Husbandry and Agricultural Chemistry, Missouri Agricultural Experiment Station and the Bureau of Animal Industry, U. S. D. A. Missouri Agricultural Experiment Station Journal Series No. 793. The investigation was supported in part by the John and Mary R. Markle Foundation.

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INTERNAL BLEEDING

Figure 9

Intra-abdominal hemorrhage was found on post-mortem examination to be the cause of death in this "bleeder".

es of these animals with other lines of the same breed; and crosses with Chester Whites, Duroc Jerseys, and Hampshires. In securing the genetic data, 24 matings involving bleeders and carriers were made and 157 pigs were farrowed. In addition 60 litters of "normal" strains from which 428 pigs were produced, contained no pigs that showed bleeding tendencies. The observations were made in 1938-40 inclusive.

A laboratory test* based on the coagulation time of decalcified citrated blood plasma was developed for distinguishing between "bleeder" and normal animals. An animal was classified as "bleeder" or "normal" on the basis of the laboratory test, prolonged bleeding time, or both. This test which has been used for diagnosing "bleeders" demonstrates that animals differ considerably in their blood coagulation times. Confidence can be placed in the test, because animals which are such severe bleeders as to have fatal hemorrhages have always been classed as severe bleeders by the result obtained when the laboratory test was used.

Results

1. *Inheritance of the abnormality.* (a) Matings of "bleeder" × "bleeder." Several matings of "bleeders" *inter se* have been made and all pigs that lived to weaning age were "bleeders."

(b) Matings of "normal" × "normal" and "normal" × "bleeder." In the

*Details of this test will be reported elsewhere.

line of breeding in which bleeding is present, normal animals mated together or to "bleeder" animals have, in some instances, produced "bleeder" as well as normal pigs.

(c) Abnormality apparently not sex-linked.

One would expect all of the boar pigs to be "bleeders" and all sow pigs normal in the mating (normal ♂ × bleeder ♀) if the male is heterogametic in swine and if the abnormality were a sex-linked recessive.

There has been no difference between the sexes in the frequency of occurrence or severity of the character. Normal boars of a line of breeding in which bleeding has not been observed × "bleeder" sows and the reciprocal mating have produced all normal pigs.

2. *Variation in age of first expression of the abnormality.* In "bleeder" pigs hemorrhage has not been observed from the umbilical cord or from notches cut in the ear soon after birth for identification purposes. Also, no laboratory method for distinguishing "bleeder" from normal pigs has demonstrated a difference between the two in early life. One pig died at four weeks of age from hemorrhage caused by an injury that removed a toe but most of the "bleeder" animals do not exhibit prolonged bleeding before they are two months of age.

3. *Variation in severity of the disease.* The abnormality increases in severity with increasing age. Such injuries as a scratch on the nose or ear frequently caused death of the mature "bleeder" animals; no "bleeder" hog has been kept to a greater age than three years because of the loss due to hemorrhage in older animals.

4. *Most frequent hemorrhagic areas.* The "bleeder" hogs had hemorrhages, sometimes fatal, from the gums, sinuses, lips, scratches on the nose or ears, and internally. Joint hemorrhages have been observed in several animals that bled to death from hemorrhage in some other region (Figures 9 and 10). Also swollen joints are frequently observed in "bleeders" (Figure 11) but these swollen joints are not always an indication of joint hemorrhage because "normal" hogs may have swollen joints. Fatal hemorrhages in the "bleeder" animals have been observed in the uterus and intestines. The bleeding from the intestines may have been caused by internal parasites. Uterine hemorrhages have followed parturition, but several "bleeder" sows delivered pigs normally and without complications.

All "bleeder" boar pigs that were castrated after they were old enough for the affliction to express itself have bled to death. Death from a wound of this size follows the operation in four or five hours, whereas, in minor hemorrhages, bleeding has been noted for six to seven

TABLE I. Breeding results involving a hemophilia-like abnormality in swine.

Type of Mating	*No. of Matings*	*No. Pigs Produced*	*Bleeders*	*Normal*	*Died Before Weaning*	*Not Tested*
Bleeder ♂ × Bleeder ♀	4	19	16	0	3	0
Carrier ♂ × Bleeder ♀	5	34	16	4	5	9
Bleeder ♂ × Known* Normal ♀	6	44	0	27†	2	15
Known* Normal ♂ × Bleeder ♀	1	7	0	7‡	0	0
Known* Known* Normal ♂ × Normal ♀	60	428	0	87§	..	341
Carrier ♂ × Carrier ♀	6	37	6	17	12	2
Bleeder ♂ × Carrier ♀	2	16	7	9	0	0

*The term "known normal" indicates that the animals were normal and from a strain in which "bleeders" have not been observed.

†The *12* males of this group were castrated at a size of 125 to 180 lbs. and none bled excessively. Bleeder animals would die if castrated at this size.

‡Four boars in this litter were castrated at three months of age and none bled excessively. The dam bled to death from a uterine hemorrhage twelve days after this litter of pigs was farrowed.

§87 boars were castrated when they weighed more than 100 lbs. and no excessive bleeding was observed. In no case has fatal hemorrhage been observed in these two lines of breeding.

BLEEDING INTO HIP JOINT
Figure 10
Autopsy showed hemorrhage in the left acetabulum or cotyloid cavity of this "bleeder".

days without fatality. It has been repeatedly observed that affected animals bleed severely after the common practice of ringing the snout.

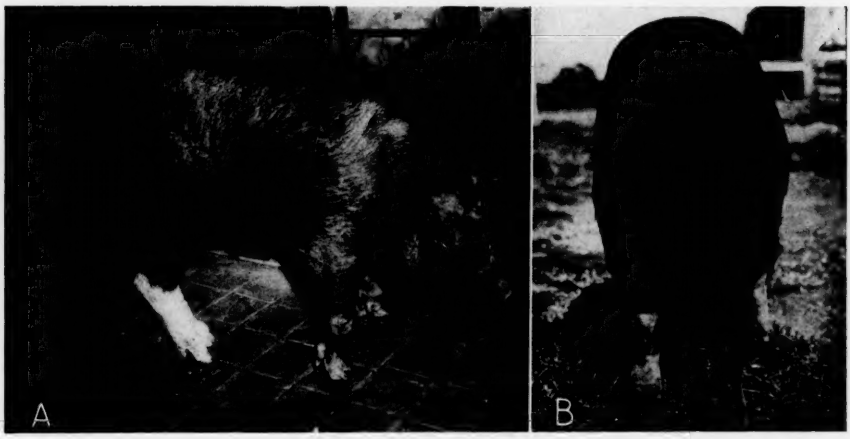
5. *Comparison of "bleeder" and normal hogs in productive characteristics.* Unless a "bleeder" is in a hemorrhagic state, no differences between "bleeder" and normal hogs is observed. Before a satisfactory test for distinguishing "bleeders" from normals was developed, several "bleeders" were kept for breeding purposes because of their superior growth rate and physical appearance. In Figure 12 a "bleeder" boar is shown to demonstrate the desirable conformation of some affected animals.

Discussion

The hemophilia-like condition in swine is similar to hemophilia in man in that fatal hemorrhage, resulting from slight injuries or occurring spontaneously, is observed in both. In both species the young animals are less severely affected than more mature ones,[11] but in swine the condition becomes worse with increasing age.

Like hemophilia in man, this condition in swine is due to a thromboplastin deficiency. All blood clotting elements have been found in normal concentrations except thromboplastin which, when added to the defective blood, causes normal coagulation.[7] That the condition resembles hemophilia more closely than purpura hemorrhagica has been shown[7] because the platelet number is no less in the "bleeders" than in the normal hogs.[8,12]

Various degrees of bleeding tendencies have been found with the laboratory diagnostic test, and several animals which are difficult to classify have been observed. Matings involving these "borderline" cases have given variable results. When severe bleeders (animals that have had hemorrhages or show severe bleeding tendencies by the laboratory test) are mated together, nothing but severe "bleeders" has been produced. When these severe "bleeders" are mated to animals of a different line of breeding in which no "bleeders" have been observed, normal animals have always been produced. Normal animals in the line of breeding in which "bleed-



SWOLLEN JOINTS
Figure 11
A—affected pig with chronic joint stiffness. Also note bleeding ear. *B*—affected pig with right rear hock joint swollen from internal hemorrhage.

ers" are present have produced both normal and "bleeder" pigs when mated together or to "bleeders." All of these results indicate the recessive nature of the abnormality. The "borderline" cases are difficult to explain. That they do not represent heterozygous animals is demonstrated by the normal offspring produced when severe "bleeders" are mated to normal animals of a line in which no "bleeders" have been observed. The results at present support the tentative conclusion that the "bleeder" abnormality is a simple recessive, but that one or more modifying factors influence the severity of the condition.

All evidence indicates that the condition is not sex-linked in swine as in the similar abnormality in man. The mating of normal boar × "bleeder" sow gave all normal pigs rather than normal females and "bleeder" males as would be expected if the condition were sex-linked.

The abnormality is economically important. A livestock feeder in Missouri bought and raised approximately 500 Hampshire and Poland China feeder pigs. When the approximately 300 boars, some of which were rather large feeder animals, were castrated, 70 were lost through hemorrhage. A veterinarian was consulted, and he reported by conversation that the animals died because the blood did not clot properly.

Two purebred breeders have reported cases which are similar to the abnormality in the herd at the Missouri Station. One breeder lost a Hampshire boar. A veterinarian performed a post mortem and found internal hemorrhage as the cause. The veterinarian suggested that the boar might have eaten some glass. The owner of the boar stated that three other animals, all sired by the old boar, died of hemorrhage. A Poland China breeder had a boar die from hemorrhage. Apparently the hemorrhage was caused by an injury in transport. Since this boar produced no offspring, genetic analysis of the condition was impossible.

Losses from castration in the inbred herd at this Station have been larger than under farm conditions because the boars have been kept to a larger size before castration to allow for more development before selections were made. Losses caused by hemorrhages that are spontaneous or follow injury have amounted to almost 20 per cent per year in the herd in which the character is present. A farmer would usually castrate his pigs at a young age. It is improbable that he would continuously use boars of the same breeding. Therefore.

A BLEEDER BOAR

Figure 12

This individual is so excellent in conformation that he would be very likely chosen for breeding purposes were it not known that he is a "bleeder".

the average farmer would have fewer losses in his herd than have occurred at this Station. This character would cause great losses to hog breeders, however, if the gene becomes widespread.

Summary and Conclusions

A hemophilia-like abnormality has been observed in one line of inbred Poland China swine.

The character first expresses itself at about two months of age, but is variable in this respect. In general the abnormality increases in severity as the animals become older.

"Bleeder" animals differ in the severity of the affliction.

It seems that the hemophilia-like character in swine is due to a recessive gene and that the variation between individuals in the severity of the abnormality is caused by modifying genes.

The character is not sex-linked as is the similar abnormality in man.

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HEREDITY AND ACCIDENT AS FACTORS IN THE PRODUCTION OF TAILLESSNESS IN THE RAT

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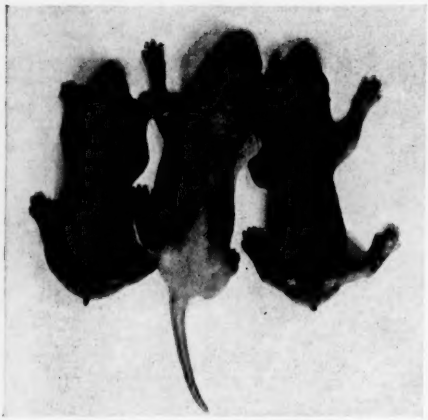
CONGENITALLY tailless forms have been reported in all of the classes of tailed vertebrates. In some cases taillessness is the result of a changed heredity and may then become characteristic of whole species, varieties, or races as in the tailless sheep, dogs, cats and chickens. In other cases occasional tailless individuals are produced by normal parents but appear not to transmit the tailless condition to their descendants. For example, among the many thousands of laboratory rats which have been observed by investigators, some nineteen sporadic cases of congenital taillessness have been recorded. Sixteen of these were reported by King[5] among some 171,000 rats observed between 1907 and 1930. King observed over 500 descendants of tailless rats but found no tailless animals among them. Even when both parents were tailless the twenty-five offspring obtained all had normal tails. She concluded that "taillessness in the rat is not a heritable but a somatic variation." Similarly, Hutt and Mydland[4] found no tailless animals among 157 F_2 descendants and 23 backcross progeny from a congenitally tailless rat.

Both the heritable and non-heritable forms of taillessness are found in the domestic fowl.[2] The former occurs in the rumpless breeds or varieties which contain a mutant gene determining absence of tail, certain sacral vertebrae and the uropygial gland; the latter are found rarely and sporadically among normal breeds of fowls and have been found with greater frequency among eggs which have been cooled early in the incubation period.[1] From these facts it was concluded that such similar phenotypes may arise when a process in early development has been rendered defective *either* by a mutant gene or by a developmental accident.

Recently we have studied cases of congenital taillessness in the rat which serve partially to bridge the gap between the so-called "hereditary" and the sporadic or accidental types. Briefly we have found tailless individuals in three different inbred families of rats, in one family with a frequency of about 1.3 per cent (13 tailless out of 1,026 observed); in another family with a frequency of about 1.6 per cent (15 tailless out of 937) and in a third family with a frequency of about 3.7 per cent (39 tailless out of 1,166 observed). In each of these families taillessness occurs more frequently than in the general population of laboratory rats, and the lines differ from each other in the frequency with which they produce tailless offspring. The tendency for tailless offspring to appear is therefore determined in part by the heredity of the family, yet the occurrence of taillessness in each family is sporadic, and, as will be shown, the tailless and normal animals within each family are probably genetically the same.

The animals observed came from three different sources. Family 1 is descended from a pair of rats sent to us in September, 1938, by Dr. Hans Grüneberg, of University College, London. This stock contained a lethal gene (Grüneberg) and was bred in order to preserve this mutation. It has now been inbred in the Genetics Laboratory for 5-6 generations (generally by brother by sister matings). In an F_3 litter two tailless females were found among nine offspring (Figure 13). The position of the tail was occupied by a small filament of skin resembling that found in tailless mice, and presumably representing, as in the mice, the remains of a tail which had been resorbed during



TAILLESS AND NORMAL RATS
Figure 13

Two tailless rats at birth with normal tailed litter mate. Each tailless rat has a small filament in the position of the tail probably representing the remains of a tail which was present in the embryo but which was resorbed before birth.

embryonic development.[3] The parents of these tailless animals were again bred and produced 10 more offspring, all normal, and when bred to other relatives, 24 normal tailed young were produced. These parents and their inbred descendants produced eleven further tailless offspring, *all females,* out of 925 progeny. The mothers of each of these tailless young were tested extensively with their brothers but only one produced more than one tailless offspring in progenies ranging from 20 to 69 young; this one female produced two tailless among 41 young. The total frequency in 7 generations was thus 13 tailless out of 1,013 or about 1.3 per cent.

Family 2, a grey hooded line derived originally from a cross of August by Copenhagen lines, was inbred (brother by sister) for 23 generations at the Crocker Institute for Cancer Research under the observation of Dr. Curtis and Dr. Dunning. A total of 1,105 young were born in this strain of which 761 were observed at weaning time. Of these 13 or about 1.7 per cent were tailless. After 1939, this line was inbred in the Genetics Laboratory where 176 young were observed at birth, of which two (both females) were tailless, or about 1.13 per cent. The total number of offspring observed was 937 of which 15 were tailless or 1.6 per cent.

Family 3, a strain of albinos derived from stock furnished by Professor H. C. Sherman of Columbia University, had been inbred brother by sister at the Crocker Institute, where in 1936 a tailless animal was observed. From that time, 11 generations of descendants of this line, all from brother by sister matings, were observed at the Crocker Institute. Out of 648 young born 527 were observed at weaning of which 21 or 3.98 per cent were tailless. This family was then bred in the Genetics Laboratory where 639 young were observed at birth of which 18 were tailless or 2.81 per cent. It is to be noted that the practice of making observations at birth instead of at weaning (one month of age) did not result in any increase in the number of tailless offspring found. This would indicate that the viability of tailless young during the first month of life is about equal to that of normal. At the Crocker Institute, the collaterals (cousins) of the first tailless animal in Family 3 were inbred brother by sister for 11 generations. At weaning 852 offspring were observed of which 21 or 2.46 per cent were tailless. Although this is a slightly lower percentage than among the direct descendants, both branches of this family were clearly marked by a higher frequency of tailless young than had ever been recorded among rats.

Because of the long continued inbreeding to which each of these families had been subjected and the consequent homozygosity attained, it seemed unlikely that any genetic difference between tailless and normal animals would be found. It was not possible to test for such differences in families 1 and 2. In family 1, all thirteen tailless animals were females and all except two failed to breed when mated with normal brothers. Each of these gave one litter of normal-tailed offspring. In family 2, three tailless females proved to be fertile and when bred to normal brothers produced respectively 30 normal; 35 normal : 1 tailless female;

and 24 normal. In family 3, however, tailless animals of both sexes were born at the Genetics Laboratory (13 females, 5 males) and all five males and two of the females proved to be fertile. The results of matings in the 11th and 12th brother by sister generations in this family are given in Table I.

TABLE I. Results of brother by sister matings in Family 3 (offspring observed at birth).

Parents	Offspring normal	Offspring tailless	Offspring total	% tailless
normal × normal	255	9	264	3.4
normal × tailless	360	9	369	2.4
tailless × tailless	16	—	16	—

It is evident that the three types of matings produce about the same results. This is the more significant since the normal animals which were bred to tailless were sibs of the tailless, and many of them had one tailless parent, so that ample opportunity for segregation of a recessive gene was given. The production of only normal tailed offspring from a cross of tailless by tailless effectively disposes of the possibility of a recessive factor for taillessness. The same result was obtained by King.[6]

It is clear that in this material no single hereditary factor determines the difference between tailless and normal individuals. It is equally clear that the likelihood that any individual will be tailless is determined by the genetic constitution of the family. It seems most reasonable to assume that the loss of the tail during embryogeny is due to the failure of some normal process, and that the frequency with which this failure occurs depends on the genotype. Taillessness in the rat is thus similar to the case of otocephaly in the guinea pig analyzed by Wright[7] and the same type of interpretation is applicable. That is, that the genetic constitution determines the threshold of a reaction which is subject to alteration by minor and more or less random accidents early in development. Defects of a specific type are therefore more likely to occur in certain genetic constitutions than in others.

It is notable that in the present case, animals with the defect (taillessness) can be shown to have the same genetic constitution as their normal sibs; and that no intermediates between normal and tailless phenotypes have been found, indicating that a reaction of an "all-or-nothing" type may precede the destruction of the tail. One may say in such cases that heredity determines how much pressure needs to be exerted on a trigger, which when tripped will produce a given effect; the actual pressure is presumably supplied by accidental irregularities in the early environment of the embryo.

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REFLECTIONS ON EVOLUTION

THIS volume* is the result of some thirty years of reflection upon the theory of evolution in general and the question of the role of natural selection in particular. It was written by an English biologist whose work gave him a phenomenally wide acquaintance with the floras of the world and their distribution. Unfortunately for science he was one of those English gentlemen for

*Willis, J. C. *The Course of Evolution.* Cambridge: at The University Press. New York: The Macmillan Company.

whom anything outside of his own orbit was of little consequence. Aside from an occasional inessential phrase the book might as well have been written in 1900 as in 1940. If Willis had any conception that the field which he discusses in such facile prose has, in the 20th Century, been the object of intense experimental attack, his book fails to show it. He presents at some length the criticisms of Natural Selection published by Fleeming Jenkin in the *North British Review* for 1867, but neither mentions nor cites any of the modern work in the same field by Wright, Fisher, or Haldane.

The author not only completely ignores the modern technical literature concerning natural selection, and most of the literature of cytogenetics; he does not even summarize the taxonomic and morphological literature critically in presenting his interesting speculations. An example (p. 49) may be given since it deals with a genus which has been investigated by the reviewer:

> A case in which mutation of this kind looks as if it might have happened in nature is that of the columbine (Aquilegia) which looks as if it might have arisen from the larkspur (Delphinium) the latter having a dorsiventral flower with one spur, the former a regular flower with five spurs. Nothing but mutation can cross the (numerical) gap between these two genera.

Unfortunately for this argument a number of intermediate forms are well known and the literature regarding them should have been immediately accessible and intelligible to a man with Willis' training. While it is true that the columbine as commonly known as a garden flower shows little indication as to how its peculiar honey spurs might have originated, there are several species in central Asia which show intermediate stages to a simple honey gland. In one of these species (sometimes put in a separate genus, Semi-aquilegia) the entire spur is nothing more than a slight glandular depression. The case has been discussed from a taxonomic viewpoint by Drummond and Hutchinson (1920)* and has even been the subject of experimental attack some years ago by Miss Schafer and the reviewer (1931).†

Willis' main conclusions are summarized in his own words (pp. 191-192):

> The process of evolution appears not to be a matter of natural selection of chance variations of adaptational value. Rather it is working upon some definite law that we do not yet comprehend. The law probably began its operations with the commencement of life, and it is carrying this on according to some definite plan * * * Mutation tends to be divergent, especially in the early stages of a family. The family, consisting probably of one genus and one species, is probably first created by a single mutation, whilst later ones are usually less marked than the first, and give rise to further genera and species * * * Chromosomal alterations are probably largely responsible for the mutations that go on." [Willis admits (p. 48) that mutations of this sort have not been seen but dismisses the objection since according to Yule.] One such mutation in fifteen to thirty years upon any small spot of the earth's surface would be sufficient to account for all the flowering plants that exist. The chance of seeing such a mutation is all but non-existent.

The bulk of the book is given over to so-called "test cases," all of which are shown to favor his hypotheses. To the student of evolution these chapters are the most valuable part of the book since they present in codified form a great variety of information with regard to speciation and distribution, though mixed with uncritical generalizations and characterized by a disregard of pertinent literature.

Willis lays much stress on the "hollow curve" of generic sizes first presented in his work on *Age and Area* (1922). As Sewall Wright has shown (1941)‡ this hollow curve of distribution is to be expected under any system of evolution in which extinction of old species is balanced by the production of new species and it is therefore without any special significance for Willis' particular theory.

Much of the evidence presented by Willis is pertinent to any real understanding of the processes of evolution. Many of the facts which he discusses (as for instance the amazing case of the Podostemaceae) are difficult to interpret in the light of any known theory. It is unfortunate for science, as well as for Willis, that he did not discuss these facts in the light of current knowledge.

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SEX-LINKED ALBINISM IN THE TURKEY

Meleagris gallopavo

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IN the spring of 1940, a near-by turkey raiser brought to us a white poult similar to that shown in Figure 15. Since it had hatched from an expensive egg that was supposed to produce a pure-bred Bronze turkey of a very desirable type, its appearance caused some dismay and a desire to know if the peculiarity were hereditary.

Studies of this poult and some of its relatives showed that the condition is caused by a sex-linked recessive gene which induces imperfect albinism.

Description

A. *Plumage*

Newly-hatched poults are not pure white, but have enough pigment in the down to cause a dingy white appearance (Figure 15). The usual irregular stripes and color pattern of Bronze poults are faintly visible in many cases. Embryos that die during the last week of incubation appear creamy in color and are easily distinguished from colored ones.

The albinotic birds raised to maturity had some pigment in the feathers. This was most dense in the regions of the feather normally colored black, and least evident in the areas where brown color is normal. In the mature turkey hen shown in Figure 14 the sub-terminal black bands of the contour feathers are faintly visible and the light barring of the primaries shows quite distinctly against the dingy white background of those feathers.

B. *Eye*

The eye of a newly-hatched albinotic poult has a pale blue-grey iris but always shows a red pupil. As the birds grow older, the color of the iris seems to darken somewhat but the pupil remains red. The decreased pigmentation of the eye is sufficient to distinguish the albinos from any dark-eyed poults or embryos.

For histological examination, the eyes of poults a day old were fixed in Bouin's fluid. Adequate penetration of this solution was ensured by cutting a hole in the wall of the eye near the corneo-scleral junction. Excised portions of the eye were cleared in toluene, embedded in paraffin, sectioned and stained with Mayer's HCl-Carmine.

Microscopical examination showed that all layers of the retina are present and apparently normal in structure in these albinos. However, melanic pigment was entirely lacking in the pecten, in the pigment epithelium of the functional retina (*i.e.* the *pars optica retinae*), and in the choroid coat (Figure 16*B*). The complete absence of melanin in these regions of the albinotic eye contrasts sharply with the abundance of dense melanin in those same parts in the eye of the normal Bronze poult (Figure 16*A*).

Anterior to the *ora serrata*, the *pars ciliaris retinae* of the albino contains some melanin but not as much as is found there in the eyes of normal poults. In the iris, the *pars iridica retinae* contains some pigment in the albinos, but the melanin seems only about half as dense as that in the irides of normal poults when sections from both types cut at the same thickness are compared (Figure 17). The iris of the albino lacks melanin in the *dilatator pupillae* muscle, which is well pigmented in the normal eye. Moreover, as might be anticipated from their bluish appearance in living birds, the irides of the albinos lack melanin on the anterior surface.

From this description, it is clear that the condition studied is not a total albinism. According to the classification of

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AN ALBINOTIC FEMALE AT EIGHT MONTHS OF AGE

Figure 14

Enough pigment is retained in the plumage to show faintly the dark bands on the contour feathers and the barring of the wing feathers that are characteristic of Bronze turkeys.

Pearson, Nettleship, and Usher,[6] it is an *imperfect albinism* because there is only partial inhibition of pigment in the eyes and feathers.

The newly-hatched albinotic poults seem to be completely blind. They make the same slow sidewise movements of the head as were observed by Hutt[7] in chicks afflicted with hereditary blindness. The poults run into obstructions and, when seeking food or water, make ill-directed pecking motions until the containers are located. Because of this handicap, the albinos are quite difficult to rear.

Genetics

A. *Evidence from the Breeder*

Because the egg from which the original albinotic poult hatched was one of a number of pedigreed eggs from trap-nested hens, it seemed probable that the turkey breeder who had supplied the eggs might be able to provide further information about albinism in his stock. Inquiry in May, 1940, elicited the information that albinism had not been seen in the breeder's turkeys prior to 1940 and that it then occurred in only one of 26 breeding pens. In this pen,

the frequency of normal and albinotic offspring in three hatches was as follows:

Dam	Progeny: Normal	Progeny: Albinotic
1	8	4
2	14	1
3	15	5
4	3	0
5	8	1
6	9	0
7	8	4
8	14	5
9	7	0
10	3	0
11	6	3
12	8	1
13	7	2
14	5	1
15	13	2
Unmarked eggs	5	2
Totals, all hens	133	31

In addition to these 31 albinotic poults that hatched, eight more were found hatched but dead from these same matings. Disregarding those eight (because the corresponding number of dead colored poults was not given), the close approach of the figures cited above to a 3:1 ratio suggested a simple recessive mutation. If it were autosomal, the expectation in progeny from the 11 hens that produced albinos would be 101 colored:34 albinos. The actual ratio was 106:29, a very good fit. If the mutation were sex-linked and viability of albinos unimpaired, the expectation would be 123 colored:41 white in the 164 offspring of the male in this pen. Again the observed numbers fitted the hypothesis fairly well, but sex of the albinos was not determined and there was no evidence that all were females, as they should have been if the mutation were sex-linked.

However, the owner reported that in other matings hens closely related to the sire or to the dams of the albinos had produced only normal Bronze poults. Because it was improbable that the male and 11 of 15 hens in one pen could carry an autosomal mutation without its cropping out in some of the 25 other breeding pens, the more likely interpretation of the data seemed to be that the new mutation was sex-linked.

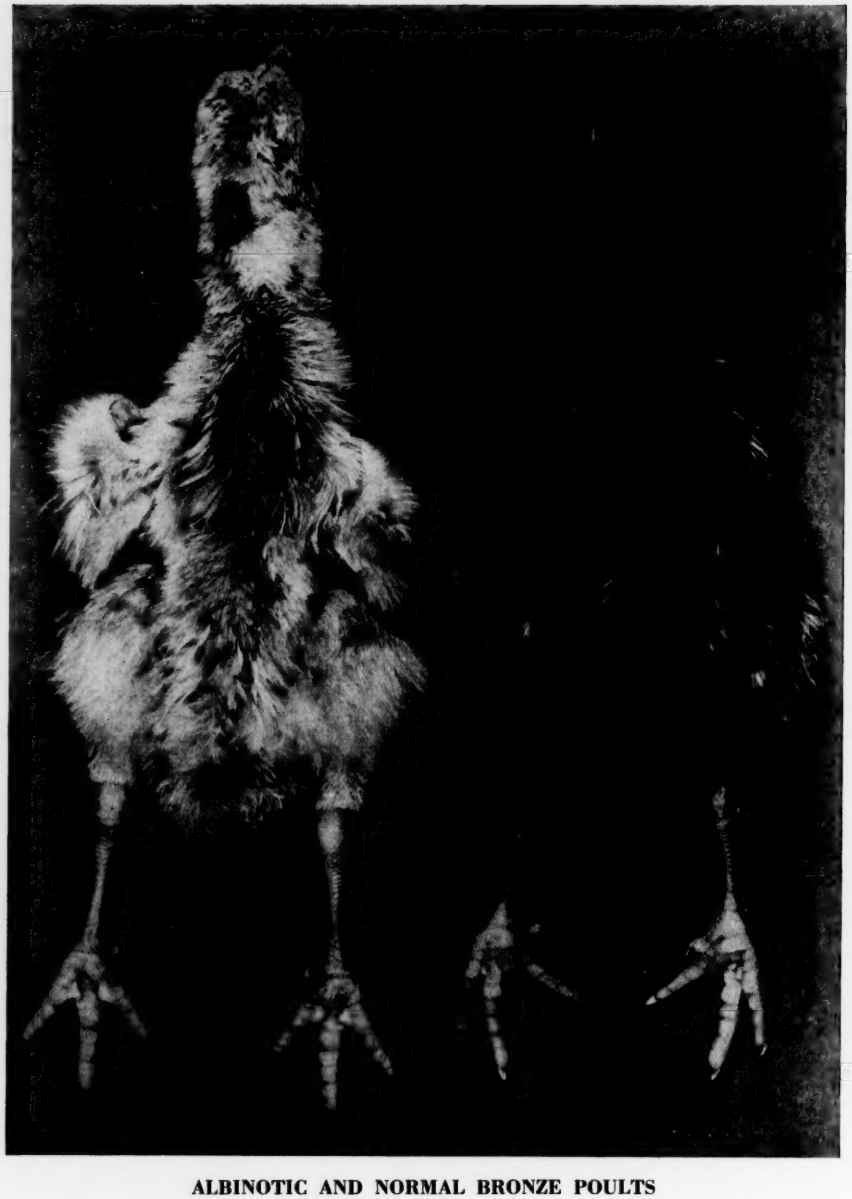
B. *Tests at Cornell*

Late in the season of 1940, 50 eggs were obtained from the breeding pen that had produced the albinos. In these, fertility was low and embryonic mortality high, with the result that only 13 poults hatched. Four of these were albinos, all of which turned out to be females. Unfortunately, only one of the albinos was raised to maturity. She did not lay until more than a year old, by which time the mode of inheritance of the character was already established. From these eggs, five toms, all normally colored Bronze, were still on hand for the breeding season of 1941. Because facilities for breeding turkeys were not available in the department, two of these males were "farmed out" to a near-by turkey raiser, who kindly arranged to put them in separate breeding pens, one male to a pen, and to let us incubate the eggs at Cornell, where the necessary examinations of dead embryos and poults could be made. A third tom went to another breeder, who hatched his own poults but supplied us with the unhatched eggs, a record of poults hatched, and the three albinotic birds from his mating.

In addition to this, the turkey breeder who brought the first albino to us made up two breeding pens, each headed by a single tom, in which all birds were normally colored and of the stock that yielded the albino, though not necessarily from the same pen as that bird. Eggs from these matings were also incubated at Cornell.

Dead embryos from these matings were examined and all those considered to be 24 days old, or more, were classified as either albinotic or colored. In younger embryos, differentiation between the two types was more difficult. Sex was determined by dissection in all late-dead embryos and in hatched albinos, but was not known for the normally colored poults because these were returned to the cooperating breeders.

Results of these matings (Table 1) showed clearly that the mutation is sex-linked. The three toms "farmed out" (Cornell 1, 2, and 3) all proved to be



ALBINOTIC AND NORMAL BRONZE POULTS

Figure 15

Both are one day old. Traces of pigment show in the down of the albino. Since this characteristic is sex-linked in the turkey, the albinos appearing in the progeny of normal-appearing "carrier" males are all females.

carriers of the gene for albinism; the two males of the original stock (O.S. 1 and 2) were not. In the combined progeny of the three carriers, including hatched poults and late-dead embryos, the ratios observed and expected were:

	Colored	*Albinos*
Observed	184	45
Expected	171.75	57.25

Application of the Chi-square test showed that the deficiency of albinos below expectation was not statistically significant ($\chi^2 = 3.48$; $p = .05$-$.10$).

The critical proof is that *all the albinos were females,* as was to be expected for a sex-linked recessive mutation. Moreover, in 34 colored poults that died during the last five days of incubation the sex ratio was 23 ♂ ♂ : 11 ♀ ♀. Since the expectation for such a character is 2 colored ♂ ♂ : 1 colored ♀ : 1 albinotic ♀, the sex ratio in the colored birds was a very close fit to that expected.

C. *Miscellanea*

The three heterozygous males showed no signs of albinism whatever. The condition may therefore be considered as completely recessive.

Symbol. To the gene for this sex-linked imperfect albinism in the turkey, the authors tentatively assign the symbol, *al.* This is done with some reservations. Because the turkey and the fowl are both members of the order Galliformes, it is desirable to use the same symbol for genes that seem to be alike in both species. We leave the symbol, *a,* clear for the autosomal albinism that may some day be found in *Meleagris gallopavo,* as it has in the domestic fowl and many other species. The symbol, *al,* has already been used by Mueller and Hutt[5] for a gene causing sex-linked imperfect albinism in *Gallus gallus.* Although, as is shown below, its effects in that species are not identical with those in the turkey, it seems probable that a common symbol for sex-linked albinism in the two species will be less confusing to geneticists than would two different ones.

Origin. It is likely that the mutation occurred only two generations earlier than that of 1940 in which it first appeared. Since albinos comprised about twenty-five per cent of the progeny of the first tom recognized as a carrier, it is fairly certain that all his primary spermatocytes must have carried the mutation and therefore that he received it from either his sire or his dam. An autosomal recessive mutation could be carried for several generations without the appearance of a homozygote, but ordinarily a sex-linked one would show in females of the first or second generation unless it were lethal to them during embryonic development.

Effects on Viability

A. *Embryos*

The records given in Table 1 show that the number of albinos dying during the last five days of incubation was a little over three times the number that hatched. This compares with the record for colored birds as follows:

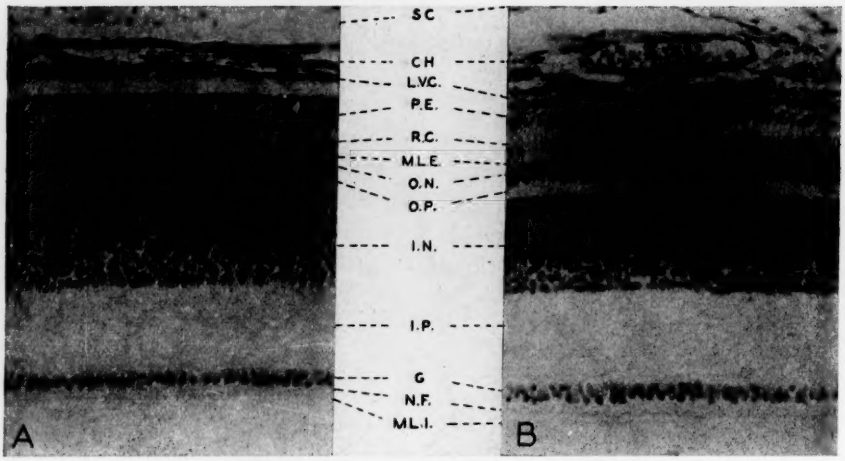
	Colored	*Albinos*
Hatched, or died late, no.	184	45
Died, 24-28 days, number	37	34
Died, 24-28 days, per cent	20.1	75.5

Under the conditions of this experiment, mortality of albinos during the last five days of incubation was nearly four times as high as in colored poults. That this happened because they were albinos, and not because they were females, is shown by the fact that, in the 34 colored embryos that died and were sexed, the proportion of females (11 ♀ ♀ : 23 ♂ ♂) was not a bit higher than the 1:2 ratio expected for this population.

It seems probable that differential mortality of albinos and normals even before 24 days of incubation would account for the slight deficiency of albinos found when only those were classified that hatched or died after 23 days of incubation. In progeny from ♂ Cornell 3, only two embryos out of 38 fertile eggs died too early to be classified, and in the remainder the ratio of 27 colored:9 albinos was exactly the 3:1 expected (Table 1). Deficiencies of albinos accompanied higher early embryonic mortality in the other two matings.

B. *Poults*

The breeder in whose stock the al-



NORMAL AND ALBINOTIC EYE STRUCTURE

Figure 16

Photomicrographs of sections through the posterior part of the eyes of day-old poults. The normal Bronze (*A*) has dense pigmentation in the choroid coat and in the pigment epithelium of the retina, but in the albino (*B*) both of these layers lack melanin entirely. The inner and outer nuclear layers in *B* appear darker than in *A*, but this results only from differences in photography. These layers contain no melanin. HCl-Carmine, 6μ, $\times$ 187.

Abbreviations:

SC — sclera
CH — choroid
L.V.C. — *lamina vitrea choroidea*
P.E. — pigment epithelium
R.C. — layer of rods and cones
M.L.E. — *membrana limitans externa*
O.N. — outer nuclear layer
O.P. — outer plexiform layer
I.N. — inner nuclear layer
I.P. — inner plexiform layer
G — layer of ganglion cells
N.F. — layer of nerve fibres
M.L.I. — *membrana limitans interna*

binos appeared stated that of 31 such poults hatched only two survived to six weeks of age. Because of their blindness most of them failed to find feed and water, and those successful in that respect were later attacked by their normal siblings, driven from the feeders, and either killed or injured so badly that they had to be destroyed.

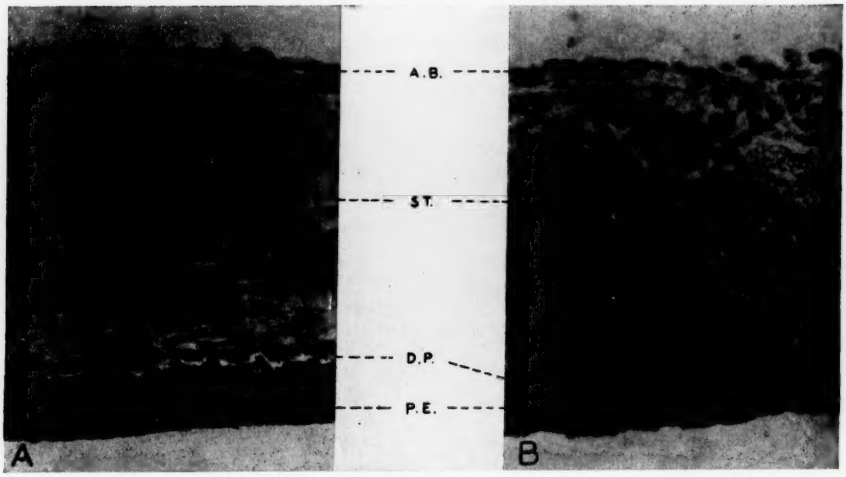
Of the four albinos hatched at Cornell in 1940, just one was raised to six months of age and that only by painstaking care. They were fed by hand for the first few days and later confined in small pens with plenty of feeders and drinking vessels. One died suddenly from an undetermined cause at five months of age.

The lone survivor finally began to lay when nearly thirteen months old. Her one colored sister, hatched at the same time and reared under identical conditions, laid when not quite eight months old.

Because it lowers the viability of embryos before hatching, and of poults after hatching, the gene, *al,* can properly be considered as a lethal gene. In nature it would undoubtedly be lethal to all females receiving it. While blindness was probably the main cause of death in albinos that hatched, the fact that the gene was semi-lethal to embryos (to which vision is presumably not so essential) suggests that other ill effects apart from blindness may have been exerted after hatching.

Comparative Genetics

Sex-linked imperfect albinism was found in the domestic fowl by Mueller and Hutt.[5] The effects of the mutation



IRIS SECTIONS

Figure 17

Photomicrographs of sections through the irides of day-old poults. In the normal Bronze (*A*), melanin is found in the anterior layer, in the *dilatator pupillae* muscle, and in the retinal layer. In albinos (*B*), melanin is confined to the retinal layer, but even there is less dense than in normal eyes. HCl-Carmine, 6μ, $\times$ 374.

Abbreviations:

A.B. — anterior border layer
ST — stroma
P.E. — pigment epithelium of the *pars iridica retinae*
D.P. — *dilatator pupillae* muscle

in that species differ from those in the albinotic turkey in three major respects, as follows:

(1) The fowl has some melanin in the pigment epithelium of the retina in the posterior half of the eye. The turkey lacks melanin in the retina posterior to the *ora serrata,* and has less pigment than the fowl in the ciliary body and iris.

(2) Viability of albinotic embryos and poults is sub-normal in the turkey, but quite unimpaired in the fowl.

(3) Vision of the albinotic fowls is apparently little affected, if at all, whereas the mutation causes complete blindness in the turkey.

Because of the difference in the distribution of melanin in the eye, it seems unlikely that the genes in the two species can be homologous, even though both are sex-linked and both induce imperfect albinism. Since they are related, one would expect the fowl and the turkey to have some genes in common. According to Sokolow, Tiniakow, and Trofimow,[7] the sex chromosome in the turkey is practically identical in size and shape with that of the fowl. Whether or not these genes for albinism occupy homologous loci in those chromosomes might possibly be determined by an inter-specific cross of the albinos, but attempts to cross the two species have been so unsuccessful that this possibility is remote. The question might be answered by finding out in each species the linkage relationships of the gene for albinism with other sex-linked genes. This has already been done for the gene in the fowl, but it cannot yet be satisfactorily undertaken for the albinism of the turkey because only one other sex-linked gene is known in that species. This, the recessive gene causing the color pattern of the Narragansett (Asmundson[1]), has not yet been proven homologous with any sex-linked gene in the fowl.

Other cases of hereditary albinism in birds are reviewed by Mueller and Hutt.[5] Sex-linked genes causing various

types of that condition are now known in four species of birds. These are the fowl, the turkey, the canary (Durham and Marryat[2]), and the budgerigar, *Melopsittacus undulatus* (Kokemüller[4]).

Economic Significance

To refute a notion, not uncommon among agriculturists, that mutations of the sort described in this paper are interesting playthings for the impractical geneticist, but nothing more, we quote verbatim one paragraph of a letter from the turkey breeder in whose stock the albinism first appeared:

> Another of our customers hatched three white poults from some eggs we shipped him, and now claims he will not be able to use any stock hatched from our eggs for fear he may have white ones. Can you tell us whether the poults coming good Bronze are apt to have offspring with any albinos? This customer is demanding full purchase price of 100 pedigreed eggs because of the white ones. About one-quarter of the eggs sent him were from Pen *A*. Anything you can tell us about this breeding problem will be appreciated.

Since these eggs sold for $2.00 each, the dismay of both buyer and seller can be readily understood. Because the mutation is sex-linked, a full answer to the problem was available in less than a year. It would have been known even in 1940 if it had then been discovered that all the albinos were females.

Only Pen *A* produced albinos. Because the mutation is sex-linked, its failure to appear in the progeny from other pens showed that none of the hens or toms in those pens carried it, nor could their offspring. In the poults from Pen *A*, the following four classes were present in approximately equal numbers except for the albinos that failed to hatch:

A. Albino females
B. Bronze females not carrying *al*
C. Bronze males not carrying *al*
D. Bronze males carrying *al*

Poults of class *A* are recognizable; those of group *B* are as desirable for breeding as any others and cannot produce albinos. This also applies to toms of class *C*, but, since they cannot be distinguished from those of group *D*, it would be best to discard all males from such a mating. If equally good toms from other pens were not available for breeding, those of class *C* could still be used provided that breeding tests were made to reveal their identity. Any Bronze females could be used for such tests. Under normal conditions, 15 or 20 eggs fertilized by one male would be ample to reveal whether or not that sire could produce albinos, especially if eggs were examined that failed to hatch.

It is doubtful if there could have been more than a dozen Bronze toms hatched from Pen *A* in the (quarter of the) 100 eggs. So far as this one transaction was concerned, the actual economic loss was small in comparison with that imagined by the buyer. Other customers may have been less perturbed, but, pending the

Table I. Records of matings proving albinism to be sex-linked.

Male	Fertile eggs	Dead embryos			Poults hatched		Total	
		Early	After 24 days				Older than 24 days	
			Colored ♂♂ ♀♀	Albinos ♀♀	Colored (Not sexed)	Albinos ♀♀	Colored	Albinos
Cornell 1	47	11	5 2	2	24	3	31	5
" 2	206	49	18 9	26	99	5	126	31
" 3	38	2	3	6	24	3	27	9
Totals	291	62	37	34	147	11	184	45
O. S. 1	175	53	28	0	83	0	111	0
O. S. 2	32	8	9	0	15	0	24	0

geneticist's solution of the case, the situation was unquestionably difficult and costly for the turkey breeder.

Summary

In a type of imperfect albinism occuring in Bronze turkeys, nearly all melanic pigment is eliminated from the plumage. In the eye, some melanin is found in the retinal portions of the ciliary body and iris, but not in the pigment epithelium of the retina in the posterior part of the eye. Affected poults are blind.

The condition is caused by a sex-linked recessive gene, *al*, which is lethal during embryonic development to about 75 per cent of the females carrying it. Most albinotic poults die within six weeks of hatching, partly because of the handicap imposed by their blindness.

Differences between sex-linked imperfect albinism in the turkey and in the fowl are pointed out and the economic significance of the defect is discussed.

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THE GREAT PEDIGREE MYSTERY

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LINKAGE RELATIONS OF SIX FACTORS IN HABROBRACON

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IN the parasitic wasp *Habrobracon juglandis* (Ashmead) females are diploid and develop from fertilized eggs, whereas the males are haploid, developing from unfertilized (parthenogenetic) eggs. Because of this, linkage work is simplified since the heterozygous F_1 females may be "bred" as virgins and the counts made of their sons.

In *Habrobracon* there is an unusually long linkage group. At the present time it is known to be at least 450 units in length extending from speckled (*sk*) to gynoid (*gy*). The present investigation deals with six factors near the middle of this group — black (*bl*), lemon (*le*), cantaloup (*c*), long (*l*), honey (*ho*), and veinless (*vl*). Since the traits have been described previously, it suffices to say that black, lemon, and honey are body colors; cantaloup is an eye color; long is an antennal character in which the antennal segments are lengthened; veinless is a wing character in which the venation is much reduced. These traits are easily recognizable and distinguishable in all combinations.

A six-point cross (Table I) and two four-point crosses (Tables II and III) are considered here. The results of other crosses will be referred to, but these data will not be presented till a later date. In Tables I-III the first column indicates the map regions in which crossing over has taken place; columns 2, 3, 4, and 5 are the alternative gametic types and their counts; column 6 gives the totals of the alternative gametic types; column 7 gives the recombination values; column 8 gives the ratios of coincidence. At the head of each table is indicated the type of cross which was made. The symbols above the horizontal line in the formula of the F_1 female indicate the P_1 maternal type; those below, the P_1 paternal type. The numbers in parentheses between these symbols designate the regions of crossing-over. The four-point crosses were made primarily to check certain assumptions concerning interference. Table III represents a summary of two identical crosses made at different times.

In order to test the linkage relations of all the factors at the same time, a six-point cross was made. Black cantaloup honey females were crossed to lemon long veinless males (Table I). The advantage of bringing all the factors into one cross in order to test their linkage relations was somewhat offset by viability differences. Lowering of viability in certain of the classes was to be expected when so many mutant types are involved, but in this case the differences are very striking when black is combined with veinless.

The ratio of coincidence for any two regions was calculated by dividing the product of crossovers involving both regions times the total count by the product of the crossovers in each of the two regions. For example, coincidence for regions 2,4 (Table I) would be all the crossovers in regions 2,4, totaling 26, times the total count, 3760, divided by all the crossovers in region 2, totaling 427, times all the crossovers in region 4, totaling 368. This gives a coincidence quotient of 0.62.

Extreme disparity of the alternative gametic types due to low viability is to be noted when black and veinless are combined. Thus the singles and triples are affected from the black cantaloup honey times lemon long veinless (Table I). Attention may be called to the six wild type quintuples of Table I. These occurred in certain related groups of fraternities and may have been diploid males in which case they should not be included.

Discussion

It has been shown in the six-point cross (Table I) that no interference ex-

ists for crossovers involving region 1. The coincidence ratios ranged from 0.93 to 1.26. In a cross lemon cantaloup honey by black long veinless (unpublished) made by Miss Frances Lannon of the University of Maine, the ratio of coincidence for region 1 ranged from 0.86 to 1.01, again showing that no interference exists for crossovers involving region 1. It has been suggested that this lack of interference is due to this region being so long. In order to test this possibility crosses were considered in which this black-lemon region could be compared with another region of similar length. In the cross of lemon females by black cantaloup honey males (Table II), the black-lemon region (23.0) and the cantaloup-honey region (22.3) are of nearly equal length, but they affect crossing-over between lemon and cantaloup very differently. Thus, the coincidence for 1,2 is 1.07 while for 2,3+4 it is only .28. In a cross lemon by black cantaloup (unpublished) coincidence for 1,2 is .78 while in a cross wild type by lemon cantaloup honey (unpublished) the coincidence for 2,3+4 is .33. Also in the cross wild type by lemon long veinless (unpublished) with the distance from lemon to veinless of about 40 units, the coincidence for 2+3,4+5 is .21.

From the foregoing it appears as though the lack of interference by region 1 is not dependent upon the length of this region, but upon some other factor. It has been pointed out in Drosophila by Graubard[1] and by Stevens,[2] that interference is absent or at least greatly reduced across the spindle attachments. Therefore, the most feasible explanation is that spindle fiber is located near lemon. This group may then correspond to one of the V-shaped chromosomes. Whether lemon lies to the right or the left of the centromere cannot be determined until another gene is found between them.

The possibility that a relatively distant region may interfere as much as or more than a nearer region lying between, is suggested by the data involving the three regions from cantaloup to veinless. Thus, in the six-point cross (Table I) coincidence for 4,5 is .97 while for 3,5 it is only .84. This is again shown in the cross cantaloup honey times long veinless (Table III) where the ratio for 4,5 is .92 while for 3,5 it is only .42 Also, in the six-point cross made by Miss Lannon the coincidence for 4,5 is .72 while for 3,5 it is .55. If the data involving regions 3,4 and 5 be summarized from the crosses mentioned here, there are, based on a total count of 9106 coincidence ratios of .41 for regions 3,4, .62 for regions 3,5 and .78 for regions 4,5. That the doubles occurring in 4,5 are subject less to interference than those in 3,5 is indicated, but far from conclusive. The actual doubles obtained in these crosses have not been sufficient numbers to warrant such a statement. It is, however, interesting that such an indication should have been obtained in four separate crosses.

TABLE I. Type of Cross - black cantaloup honey x lemon long veinless.

F_1 bl + (1) le + (2) + o (3) + 1 (4) + ho (5) + vl

Regions	F_2 males			Totals	Crossover values(%)	Ratios of Coincidence
0	bl.c.ho	912	le.l.vl.	741	1653	
1	bl.le.l.vl.	42	c.ho.	546	588	27.9
2	bl.l.vl.	82	le.c.ho.	193	245	11.3
3	bl.c.l.vl.	58	le.ho.	299	357	15.7
4	bl.c.vl.	64	le.l.ho.	166	228	9.8
5	bl.c.ho.vl.	36	le.l.	106	142	7.9
1,2	bl.le.c.ho.	59	l.vl.	52	111	1.1o
1,3	bl.le.ho.	75	c.l.vl.	83	158	1.11
1,4	bl.le.l.ho.	20	c.vl.	55	75	.95
1,5	bl.le.l.	34	c.ho.vl.	38	72	1.26
2,3	bl.ho.	9	le.c.l.vl.	1	10	.31
2,4	bl.l.ho.	3	le.c.vl.	7	10	.62
2,5	bl.l.	12	le.c.ho.vl.	8	20	1.25
3,4	bl.c.l.ho.	2	le.vl.	14	16	.73
3,5	bl.c.l.	8	le.ho.vl.	5	13	.84
4,5	bl.c.	2	le.l.ho.vl.	4	6	.97
1,2,3	bl.le.c.l.vl.	0	ho.	2	2	
1,2,4	bl.le.c.vl.	2	l.ho.	3	5	
1,2,5	bl.le.c.ho.vl.	0	l.	13	13	
1,3,4	bl.le.vl.	3	c.l.ho.	3	6	
1,3,5	bl.le.ho.vl.	2	c.l.	6	8	
1,4,5	bl.le.l.ho.vl.	0	c.	2	2	
2,3,4	bl.vl.	1	le.c.l.ho.	0	1	
2,3,5	bl.ho.vl.	0	le.c.l.	0	0	
2,4,5	bl.l.ho.vl.	0	le.c.	1	1	
3,4,5	bl.c.l.ho.vl.	0	le.	9	9	
1,2,3,4	bl.le.c.l.ho.	0	vl.	1	1	
1,2,3,5	bl.le.c.l.	0	ho.vl.	0	0	
1,2,4,5	bl.le.c.	1	l.ho.vl.	0	1	
1,3,4,5	bl.le.	1	c.l.ho.vl.	1	2	
2,3,4,5	bl.	1	le.c.l.ho.vl.	0	1	
1,2,3,4,5	bl.le.c.l.ho.vl.	0	+	6	6	
					3760	

TABLE II. Type of Cross - lemon x black cantaloup honey.

F_1 + le + + o (3 4) + ho + bl (1) + (2)

Map Region	F_2 males			Totals	Crossover values(%)	Ratios of Coincidence
0	le.	579	bl.c.ho.	440	1019	
1	bl.le	124	c.ho	189	313	23.0
2		82	le.c.ho.	98	179	12.6
3 4	bl.c.	166	le.ho.	180	346	22.3
1,2	bl.le.c.ho.	18	+	41	59	1.07
1,3+4	bl.le.ho	35	c.	54	89	.89
2,3+4	bl.ho	10	le.c.	3	13	.28
1,2,3+4	bl.le.c.	1	ho.	2	3	
					2020	

TABLE III. Type of Cross - cantaloup honey x long veinless.

F_1 o + ho + + (3) 1 (4) + (5) vl

Map Region	F_2 males				Totals	Crossover values(%)	Ratios of Coincidence
0	c.ho	883	l.vl.	577	1460		
3	c.l.vl	166	ho.	94	259	13.3	
4	c.vl.	99	l.ho.	85	174	9.4	
5	c.ho.vl.	73	l.	57	130	7.2	
3,4	c.l.ho.	2	vl.	2	4		.53
3,5	c.l	2	ho.vl.	4	6		.42
4,5	c.	7	l.ho.vl.	2	9		.92
3,4,5	+	3	c.l.ho.vl.	0	3		
					1043		

The author wishes to thank Dr. P. W. Whiting for suggesting this problem and for giving constant assistance throughout the course of this investigattion.

Summary

1. Two four-point and one six-point crosses were made involving the factors black, lemon, cantaloup, long, honey, and veinless.

2. Crossover percentages in these experiments range from 23.0 to 27.9 for black and lemon, from 11.3 to 12.5 for lemon and cantaloup, from 13.3 to 15.7 for cantaloup and long, from 9.4 to 9.8 for long and honey, and from 7.2 to 7.9 for honey and veinless.

3. Evidence is presented to show that there is no interference by crossing-over in the black-lemon region with any region to the right of lemon. It is suggested that the spindle fiber attachment may be near lemon.

4. It is indicated that double crossovers occurring in the cantaloup-long, honey-veinless regions have a lower ratio of coincidence than do the doubles occurring in the long-honey, honey-veinless regions, or that in this case interference is greater with a distant than with a nearer region.

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◆

Aping the Man

(*Continued from page* 44)

cant fact is that a widely distributed and apparently ancient group of Negroids (the pygmies) — physically the most distinctive — has been stunted simultaneously in body size and culture." Bushmen and Australian "Black Fellows" furnish similar examples. Even within modern nations, such as France and Germany, certain groups are different from their neighbors in a way that suggests an intimate relation between bodily form and culture.

In any book by Professor Hooton we expect not merely two personalities, but three. He is primarily a good scientist, but he is also an inveterate humorist, and an ardent propagandist. Eugenics is the theme of his warmest propaganda. Nevertheless, in spite of many good words for eugenics, he does not point out the eugenic significance of his findings except by implication. Nor does he give a complete treatment of his subject. Purposely, or otherwise, he leaves the reader much interested, but almost annoyed because he stops at the most critical point. Here is what he says about it:

"It may occur to you that I have been speaking with confidence about the linking of the individually variable human organisms with their equally variable functional possibilities, but have cited little concrete factual data to validate my contention. (This is true.) The difficulty is not paucity of evidence but a veritable embarrassment of riches. The interrelated facts, however, are at present so sporadic that an enumeration of them would be time consuming and more confusing than clarifying to lay readers."

We wish that Professor Hooton, with his scientific judgment, genial wit, and pleasant style would write a well balanced book in which he fully develops all sides of the problem of bodily form and human behavior.

Ellsworth Huntington
Yale University

"HEREDITY" LANTERN SLIDES

MAN

16-397. Gregor Mendel's Handwriting — First Page of "Studies of Plant Hybrids".
17-435. Merton House—Home of William Bateson.
21-1-Cover. The "Fuchsia-Picture" of Mendel.
21-No. 2-Cover. Francis Galton.
26-158. Ishmael Family—Six Generations of a Degenerate Family.
26-159. Roosevelt Family—Pedigree Chart of a Superior Family.
26-292. Dr. and Mrs. Hugo de Vries.
15-330. Fused Finger Joints.
17-393. Short Legs—Inherited Achondroplasia.
20-75. Extra Fingers—Recessive Polydactylism.
20-74. Chart Showing Inheritance of Polydactylism.
20-352. A Folding Man — Hereditary Absence of Clavicles.
22-104. Crooked Arms and Legs—Hereditary Bone Fragility.
23-307. Five Generations of Glaucoma.
23-425. Inherited Doublejointedness in the Thumb.
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22-40. Corresponding Fingerprints of 3 Pairs of Identical Twins.
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23-5. Identical Twins Reared Apart—Mary and Mabel as Babies.
23-6. Identical Twins (Mary and Mabel) at Two Years—Reared Apart.
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23-9. Palm Prints of an Identical Twin (Mabel).
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25-446. Inherited Chest Depression—Koilosternia.
25-483. Sex-Linked Toothlessness.
25-338. Fifty-fifty Segregation for Woolly Hair Mutation.
26-143. Extra Toes—Inherited Polydactylism.
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10-409. Typical Identical Twins.
20-50. Identical Twins Reared Apart.
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20-155. City Boy and Country Boy—Identical Twins Reared Apart.
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PLANT

15-204. Capsules of Globe Daturas.
17-372. Awn of Manchuria (Rough) Barley.
17-373. Awn of Lion (Smooth) Barley.
18-128. Adaptive Cupped Tendrils of a Plant.
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18-382. Leaves of Live Oak × Post Oak Hybrids.
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19-419. "Fatback" and "Razorback" Honey Locust Pods.
20-24. Haploid and Dwarf Diploid Tomato Plants.
20-25. Haploid and Normal Tomato Plants.
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20-206. Flax Hybrid Produced by Artificial Nutrition.
22-368, 370. Plant Patents No. 2 and No. 3.
24-131. Radium-Induced "Ridged" Tomato.
24-133. Chimeral Radium-Treated Tomato Leaves.
24-134. Radium-Induced Virescent-White Tomato Seedlings.
24-368. Gene Mutation Chimera in Delphinium.
26-137. Ears of Maize, Relatives, and Hybrids.
25-448. Fasciated Sugar Beet.

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AMERICAN GENETIC ASSOCIATION

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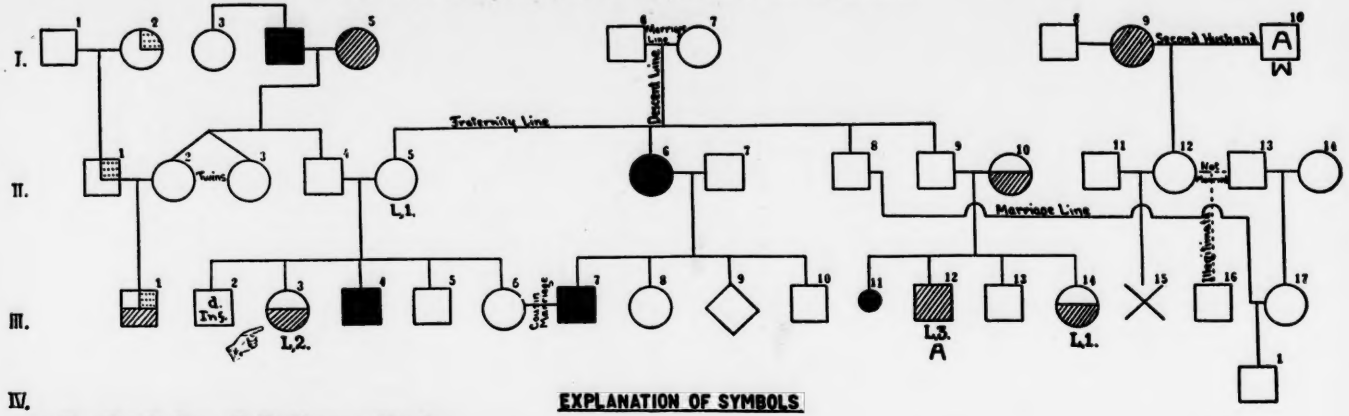
SAMPLE PEDIGREE CHART SHOWING THE MANNER OF CONSTRUCTION, AND THE USE OF STANDARD AND SPECIAL SYMBOLS.



EXPLANATION OF SYMBOLS

(a). **Standard for all Pedigree Charts:**

□=Male; ○=Female; ◇=Sex unknown; ●=Still-birth or Miscarriage; X=Children — number and sex unknown; ○○=Twins; d. Inf.=Died in Infancy; ☞ Points to the Propositus or central figure in the pedigree.

□—○=Parents.

□○□○○=Children.

Roman figures to the left indicate generations; Arabic figures locate individuals, (thus III. 7 is the young man in the third generation who married his cousin).

The following letters, placed in or around the individual's pedigree symbol, are standard for certain traits: A, alcoholic; B, blind; D, deaf; E, epileptic; F, feebleminded; I, insane; M, migraineous; N, normal in reference to traits under consideration; Ne, neurotic; P, paralytic; Sx, sexually immoral; S, syphilitic; T, tuberculous; W, wanderer.

(b). **Especially devised or selected for this particular pedigree-chart:**

■ ● = Successful leaders in politics. ⓤ Ⓤ = Extra thumb on right hand. L.1. = Highly successful author. L.3. = Little or no ability in literary efforts. ▨ ◍ = Superior in vocal music. ▨ ◍ = Medium attainment in vocal music.

To fit the particular family and traits (whether physical, mental or temperamental; good or bad) under consideration, invent special symbols, or select special letters (in addition to those standardized under (a) above) to be placed within or near the particular individual's pedigree symbol, to indicate particular traits and their degree of development.

Prepared by the International Commission of Eugenics. See *Journal of Heredity* for August 1926. Additional copies may be obtained from A.G.A.